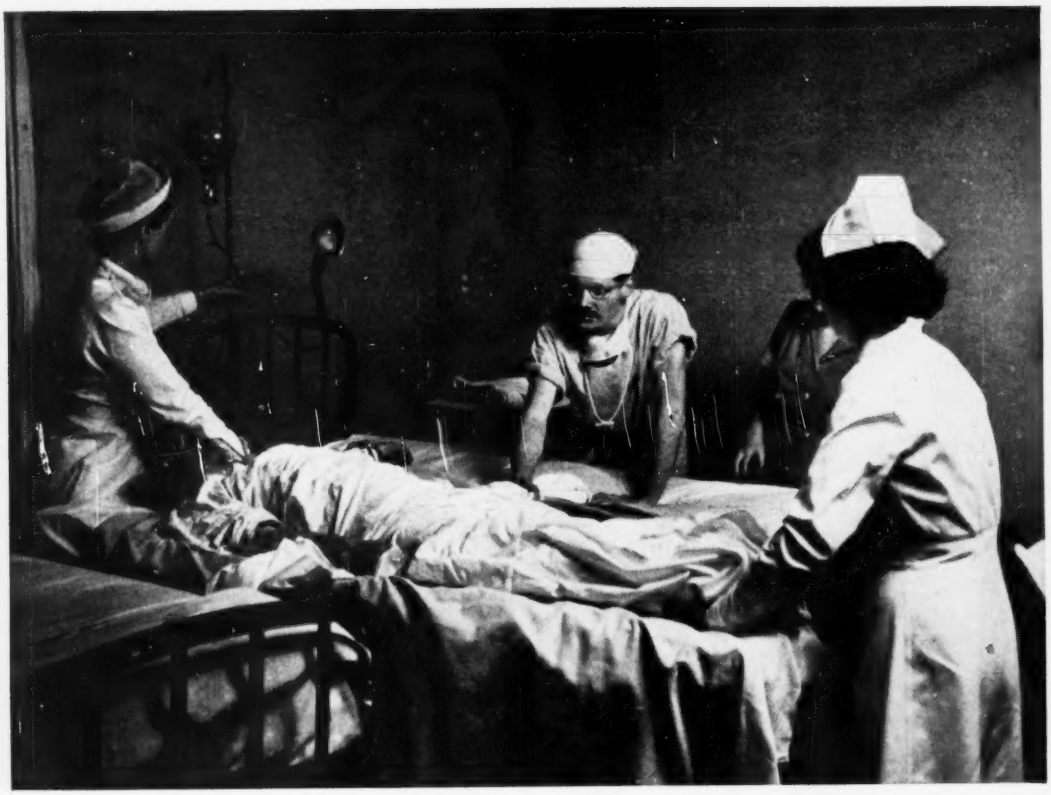
THE American Journal OF Gastroenterology

VOL. 22, NO. 5 NOVEMBER, 1954

Second Annual Convention
Chicago, Ill.
24, 25, 26 October 1955

Official Publication
AMERICAN COLLEGE
OF GASTROENTEROLOGY



when his need is greatest... **postoperatively**

Severe or rapid depletion of water-soluble vitamins is effectively and optimally countered by ASF – Anti-Stress Formula. Fulfilling the recommendations of the Committee on Therapeutic Nutrition, National Research Council, ASF supplies the critical vitamin needs of the patient during periods of physiological stress.

Each ASF Capsule contains:

Thiamine Mononitrate	10 mg.
Riboflavin	10 mg.
Niacinamide	100 mg.
Pyridoxine Hydrochloride	2 mg.
Calcium Pantothenate	20 mg.
Ascorbic Acid	300 mg.
Vitamin B_{12} Activity	4 mcg.
Folic Acid	1.5 mg.
Menadione (vitamin K analog)	2 mg.

Dosage: 2 capsules daily in severe pathologic conditions; 1 capsule daily when convalescence is established.

Supplied: bottles of 30 and 100.

**Trademark*

stress New **ASF*** *(Anti-Stress Formula)*

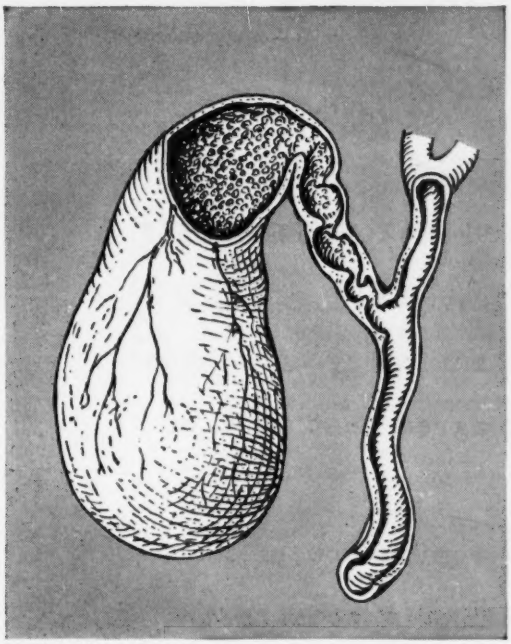
BASIC PHARMACEUTICALS FOR NEEDS BASIC TO MEDICINE

536 Lake Shore Drive, Chicago 11, Illinois

Rx POERIG

By increasing bile secretion with Ketochol® and controlling sphincter of Oddi spasticity with Pavatrine®, a free flow of bile is instituted with resultant symptomatic improvement.

Conservative, Effective Medical Management of Chronic Gallbladder Disease



Gallbladder and ducts.

The ketocholanic acids in Ketochol stimulate the flow of hepatic bile and flush the bile ducts. Antispasmodic medication, as provided in Pavatrine, diminishes gastrointestinal irritability and, by relaxing the sphincter of Oddi, effectively reduces symptoms of colic. This therapeutic program offers rational, conservative therapy in gallbladder dysfunction.

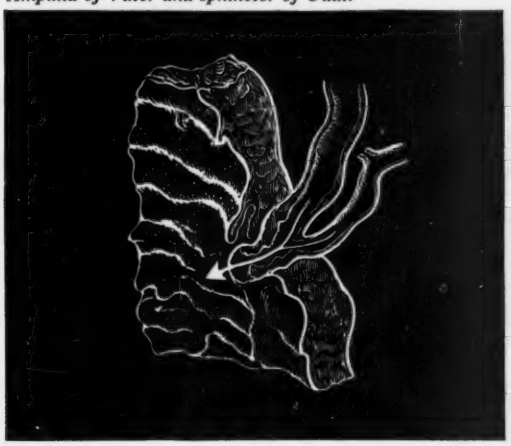
That the four bile acids present in Ketochol relieve biliary stasis is even more definitely proved by their use in the diagnosis of nonvisualized gallbladders. After the administration of Ketochol, repeat cholecystograms permitted[1] correct diagnoses.

In conjunction with the foregoing medication, proper diet, adjusted intake of milk and cream and mental relaxation are important.

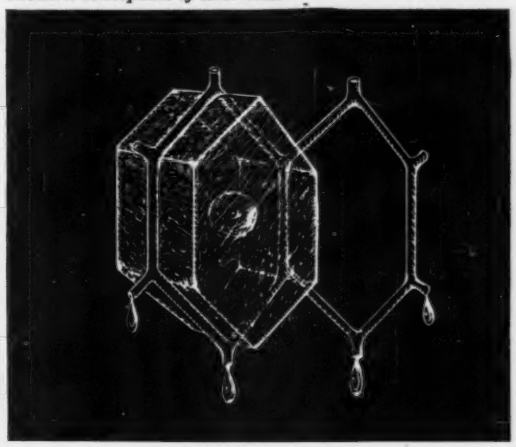
The average dose of Ketochol is one tablet three times daily with or following meals. The average dose of Pavatrine or Pavatrine with Phenobarbital is one or two tablets three or four times daily as needed. G. D. Searle & Co., Research in the Service of Medicine.

1. Berg, A. M., and Hamilton, J. E.: A Method to Improve Roentgen Diagnosis of Biliary Diseases with Bile Acids, Surgery *32*:948 (Dec.) 1952.

Ampulla of Vater and sphincter of Oddi.



Modern conception of liver cell.



Speaking of antacids—

WHICH DO YOU PRESCRIBE?

Regardless of which antacid you've been using, we believe you'll agree that most of them are rather good.

Still, we'd like to remind you of Syntrogel® 'Roche'...because it acts fast (in a matter of seconds) and long (often for hours). For patients with heartburn or too much stomach acid, Syntrogel is really worth trying.

VOL. 22, NO. 5 NOVEMBER, 1954

THE American Journal OF Gastroenterology

(FORMERLY THE REVIEW OF GASTROENTEROLOGY)

The Pioneer Journal of Gastroenterology, Proctology and Allied Subjects in the United States and Canada

contents:

Owned and published monthly by the American College of Gastroenterology, Inc. Business Office: 33 West 60th St., New York 23, N. Y. Editorial Office: 146 Central Park West, New York 23, N. Y. Subscription rate, U. S. and possessions: One year $6.00, two years $10.00 (foreign $8.00, $14.00). Single copy: $.75. Reentered as second class matter at the Post Office at New York, N. Y., under the act of March 3, 1879.

Index to Advertisers

OFFICIAL PUBLICATION

of the

AMERICAN COLLEGE OF GASTROENTEROLOGY

33 West 60th Street, New York 23, N. Y.

Editorial Office, 146 Central Park West, New York 23, N. Y.

SAMUEL WEISS, *Editor-in-Chief*

Publication Office, 33 West 60th Street, New York 23, N. Y.

DANIEL WEISS, *Managing Editor*

STEVEN K. HERLITZ, *Advertising Manager*

Contributions: Articles are accepted for publication on condition that they are contributed solely to THE AMERICAN JOURNAL OF GASTROENTEROLOGY. Manuscripts should be typewritten double-spaced and submitted in two copies. Footnotes and bibliographies should conform to the style recommended by the American Medical Association, illustrations and diagrams should carry suitable lettering and explanations, be mounted on separate pages and have the name of the author on each page. Four illustrations per article are allowed without cost to the author.

Reviews: THE AMERICAN JOURNAL OF GASTROENTEROLOGY will review monographs and books dealing with gastroenterology or allied subjects. It may be impossible to review all material sent. However, an acknowledgement will be made in the Department of Reviews.

The editors and publishers are not responsible for individual opinions expressed by their contributors, nor for those given under current literature.

Reprints: A price list and order blank for reprints will be sent to each contributor before the journal is issued.

Subscription price: U.S. and possessions: one year, $6.00, two years, $10.00. Elsewhere, $8.00, $14.00. Single copy $.75. Members of the American College of Gastroenterology receive the JOURNAL as part of their membership.

Change of Address: Notify publishers promptly of change of address. Notices should give both old and new addresses.

prescribe

TRICREAMALATE®

for the patient
with an
"eccentric"
ulcer

Roentgenographically, their peptic ulcers may appear about the same. But the "eccentric" ulcer patients bleed easily, have frequent flare-ups, and suffer from persistent pain. Tricreamalate in such a case often gives relief and even helps to avoid surgery.

Tricreamalate (*reactive* aluminum hydroxide *plus* hydrated magnesium trisilicate) stops pain fast — prevents recurrences — helps to control bleeding — is nonconstipating — prolongs buffering action. Liquid and tablets for PEPTIC ULCER and GASTRIC HYPERACIDITY.

Winthrop-Stearns INC.

New York 18, N. Y. • *Windsor, Ont.*

the most potent

multiple vitamin

you can prescribe

'Theracebrin'

(Pan-Vitamins, Therapeutic, Lilly)

especially in major surgerv,

severe burns, infectious hepatitis

EACH GELSEAL CONTAINS:

Thiamin Chloride	15 mg.
Riboflavin	10 mg.
Pyridoxine Hydrochloride	3 mg.
Pantothenic Acid (as Calcium Pantothenate)	20 mg.
Nicotinamide	150 mg.
Vitamin B_{12} (Activity Equivalent)	10 mcg.
Folic Acid	0.33 mg.
Ascorbic Acid	150 mg.
Distilled Tocopherols, Natural Type	25 mg.
Vitamin A Synthetic	25,000 units
Vitamin D Synthetic	1,500 units

Supplied in 30's, 100's, and 500's.

Dosage: One or more daily.

ELI LILLY AND COMPANY, INDIANAPOLIS 6, INDIANA, U.S.A.

Ulcer pain and other symptoms disappear rapidly"[1]

PRANTAL®

anticholinergic

with outstanding freedom

from side effects[2]

Individualized therapy of peptic ulcer, pylorospasm, gastric hyperacidity and hypermotility, and chronic hypertrophic gastritis.

For easily adjusted dosage . PRANTAL Tablets
For eight hours' relief
with single dose . . . . . PRANTAL REPETABS
For acute episodes . . . . . PRANTAL Injection

1. Hoffmann, C. R.: Am. Pract. & Digest Treat. *4*:464, 1953.
2. Riese, J. A.: Am. J. Digest. Dis. *21*:81, 1954.

PRANTAL® Methylsulfate, brand of diphemanil methylsulfate.

REPETABS,® Repeat Action Tablets.

Schering

PRANTAL

SCHERING

Because it is widely known throughout the world and has demonstrated its effectiveness in rapidly controlling the great majority of common infections, this broad-spectrum antibiotic is prescribed with certainty by physicians the world over.

terra cognita Terra

Supplied in the many convenient forms required in the practice of modern medicine: Capsules, Tablets (sugar coated), Pediatric Drops, Oral Suspension, Intravenous, Intramuscular, Ophthalmic (for solution), Ophthalmic Ointment, Ointment (topical), Vaginal Tablets, Troches, Otic, Nasal, Aerosol, Soluble Tablets and Topical Powder.

PROVED

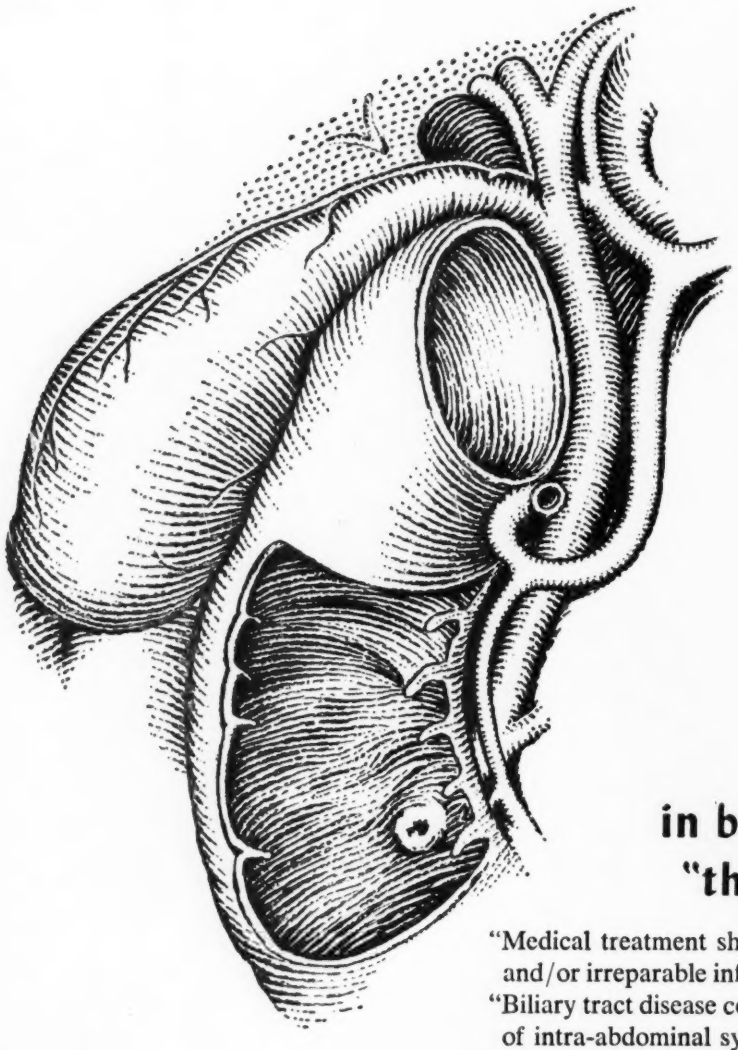
mycin®

BRAND OF OXYTETRACYCLINE

rapid absorption
wide distribution
prompt response
excellent toleration

Pfizer

PFIZER LABORATORIES, *Brooklyn 6, N. Y.*
DIVISION, CHAS. PFIZER & CO., INC.



in biliary stasis... "therapeutic bile"

"Medical treatment should be tried before stones and/or irreparable inflammation have occurred."[1]

"Biliary tract disease comprises an important cause of intra-abdominal syndromes. . . . Medical management is the accepted treatment for functional disorders."[2]

Decholin® and Decholin Sodium®

(dehydrocholic acid, *Ames*) (sodium dehydrocholate, *Ames*)

". . . increase the volume output of a bile of relatively high water content and low viscosity."[3]

Decholin Tablets, 3¾ gr. (0.25 Gm.), bottles of 100, 500, 1000 and 5000. ***Decholin Sodium***, 20% aqueous solution, ampuls of 3 cc., 5 cc. and 10 cc.; boxes of 3, 20 and 100.

1. Segal, H.: Postgrad. Med. *13*:81, 1953. 2. O'Brien, G. F., and Schweitzer, I. L.: M. Clin. North America *37*:155, 1953. 3. Beckman, H.: Pharmacology in Clinical Practice, Philadelphia, W. B. Saunders Company, 1952, p. 361.

AMES COMPANY, INC.
Elkhart, Indiana
Ames Company of Canada, Ltd., Toronto

93754

THE American Journal OF Gastroenterology

A monthly journal of Gastroenterology, Proctology and Allied Subjects
(FORMERLY THE REVIEW OF GASTROENTEROLOGY)

VOLUME 22 NOVEMBER, 1954 NUMBER 5

OBSERVATIONS ON THE PATHOLOGY AND THERAPY IN BILIARY TRACT SURGERY*

ABRAHAM AYALA GONZALEZ, M.D., F.A.C.S.†
Mexico City, D.F.

A careful analysis of phenomena which persist or appear after surgery on the biliary tract may lead to certain conclusions that might alter our present knowledge. I am referring particularly to the so-called postcholecystectomy syndrome. This latter nomenclature should be discarded because it is vague in meaning and confuses stages with manifestations. It comprises manifold syndromes of several causes, occasionally confounding conditions without a precise relationship to the operative intervention. On occasion it may mimic a hypostenic dyspepsia and again, it may resemble a crisis similar to those to be treated surgically. They have nothing in common except their appearance or persistence after cholecystectomy but it may disturb the postoperative course early or even years later. The physician may be impressed by the discovery of gallstones and he may interpret the entire pathology of the patient upon this finding forgetting though that there may, in addition, be present a diaphragmatic hernia, peptic ulcer or any other process which has no connection to the cholecystitis and which, in fact, may be entirely responsible for the postoperative manifestations. This error may be committed by the clinician and the surgeon as well who, after confirming his diagnosis of gallstones, is content with the treatment of the gallbladder while leaving the operative diagnosis incomplete because of failure of performing a comprehensive exploration. The pancreas, which often is responsible for the preoperative manifestations, continues unaffected after surgery because the surgeon was content merely with extirpation of the gallbladder. A preoperative, accurate diagnosis is indispensable and insures a satisfactory treatment which will avoid unnecessary suffering on the part of the patient, as well as remove an everlasting distrust by those not cured by the operation. Our contribution must be efficient in order that the postoperative course will not be obscured by the

*Revised by Dr. Michael W. Shutkin, Milwaukee, Wisc.

†Pres. Mexican Association of Gastroenterology, Member of the Mexican Academies of Medicine and Surgery, Head of the Dept. of Gastroenterology of the General Hosp., Mexico City, D. F.

misnamed postcholecystectomy syndrome that occasionally is really a precholecystectomy syndrome. Because of these considerations I prefer to include in my present discussion both organic and functional manifestations as well as the complications and accidents.

A prologue to a French publication involving the results of these operations warns the beginner not to be discouraged in the analysis of these phenomena. Bockus in his book "Gastroenterology for Graduates" recommends that surgery of the biliary tract should be the last stage for those being trained in abdominal surgery and that the treatment of a pathological state of the biliary tract should be performed by a highly experienced surgeon. Our wards should not contain numerous patients subjected to gallbladder surgery who continue to suffer from stenosis of the ducts and end their lives as invalids. The advice of Bockus reflects his experience as a gastroenterologist who sees this problem following surgery by inexperienced hands. In a former paper I reported that in my Gastroenterological

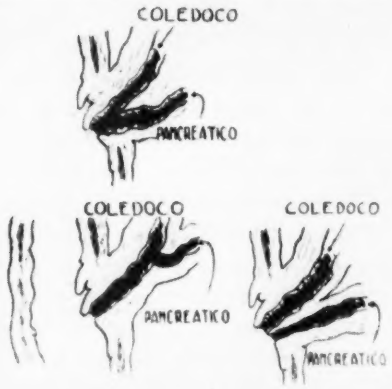


Fig. 1—Top. Most Common form. Both ducts end in a small common cavity.
Lower right. A common duct for both which leads into the papilla.
Lower left. The common cavity is absent, and they discharge simultaneously into the papilla.

Service at the General Hospital, I had occasionally under my supervision, up to ten patients previously operated upon, who returned because of postoperative manifestations, some appearing early, others unchanged and those in which they appeared in later life. When the clinician has failed to realize, after his preoperative examination, that his patient not only suffers from surgical cholecystopathy but also from chronic pancreatitis or ulcer of the duodenum, chronic hepatitis or a diaphragmatic hernia, the surgeon is privileged to perform a complete surgical exploration and thereby attempt to correct the proper pathological state. The surgeon must contribute skill and speed but this must not replace his complete knowledge of the affliction nor permit the risk of removing the gallbladder hurriedly, thereby overlooking stones in the common duct or pancreatitis which narrows the ampulla of Vater. We are able to state in a general way that patients with a calculus cholecystitis respond better than those

with nonlithic cholecystitis; that about 90 per cent of the calculous patients operated upon who have only a cholecystitis turn out satisfactorily while only 50 to 60 per cent of the nonlithic cases remain completely asymptomatic.

The preceding considerations, and those I shall enumerate later, are based on results I obtained by analyzing my working material of seven years (1945-1951) on patients in the "Servicio de Gastroenterologia" of the General Hospital and on private patients operated on both at the "Gaston Melo Wards" and at the American-British Cowdray Hospital.

It is possible that this analysis of my surgical cases is not as satisfactory as might be desired; however, I believe it is useful and moreover, it reveals my personal experiences. The greater number of patients are from the General Hospital, but I believe it convenient to add my private patients from the "Gaston Melo Wards" and from the American-British Cowdray Hospital, all of which were observed during the same period. These patients I have treated myself without reference to those operated on by other surgeons.

Operations performed on the biliary tracts between 1945-1951 (seven years).

TABLE I

At the "Servicio de Gastroenterologia of the General Hospital	268
At the "Gaston Melo Wards"	28
At the American-British Cowdray Hospital	76
Total	372

This figure indicates the number of operations practiced on 364 patients. The 364 patients under consideration were subjected to 372 operations because in some instances, two and three operations were performed. For accurate interpretation of these figures it is necessary to account for the fact that some patients had been operated upon previously by other surgeons.

Sex

		Percentage of Cases	
Women	292	80.2	Proportions:
Men	72	19.8	4 women to 1 man

Age

(Age according to data of 348 patients)
Minimum age: 11 years
Maximum age: 86 years

Age	Cases	Percentage	
11 - 19	4	1.1	
20 - 29	51	14.6	
30 - 39	70	22.7	
40 - 49	89	25.5	(68.9)
50 - 59	72	20.7	
60 - 69	50	14.3	
70 - 79	2	0.5	
86	1	0.3	

The female patients were in proportion of four to each male patient, in accordance with statements made before.

Considering the nature of this publication, it is not convenient to discuss now the factors that explain the results of these statistics. Though the figures obtained do not reveal the actual picture they, nevertheless, give a very close indication of what is happening.

The age of 16 patients is not mentioned because of an omission in the clinical history. With respect to age, it is seen that the greatest numbers presented are in the ages between 30 to 59 years, which is after the third decade of life, an increase for patients from 40 to 49 years and more or less equal for those from 30 to 39 and 50 to 59 years. The proportion is decidedly lessened for youths and for the aged.

The exact onset of the disease in 31 of the registered patients could be established effecting the necessary reduction of the total cases involved to 333. The clinical age of the disease in this series had a minimum period of four days, a maximum of 45 years, with an average of 7.8 years. The duration of the disease

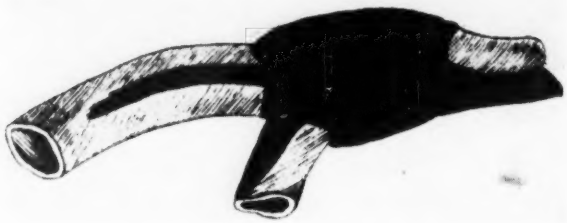


Fig. 2

is generally an approximate estimation since the patient frequently takes only into consideration that period which notably alters his health. The approximate average of eight years is not an absolute figure since many factors, such as environment, patient intelligence and the perspective of the physician may all modify this conclusion. The same considerations may be evaluated with respect to the variable behavior observed in acute and chronic noncalculus cholecystitis.

TABLE II

DOMINANT SYMPTOMATOLOGY IN 334 PATIENTS

	Cases	Percentage
Painful crisis without jaundice	179	53.5
Painful crisis with jaundice	117	35.0
Asymptomatics	15	4.5
Dyspeptic biliary syndrome	14	4.1
Obstructive jaundice	7	2.0
Biliary ileus	2	0.5
Total	334	99.7

The nonicteric group predominated to the degree of 53.5 per cent while 35 per cent had painful crises with jaundice. Among the asymptomatic cases, diseased gallbladders were found with surgical intervention when indications for surgery were based upon gastric or duodenal afflictions. In others with minimal symptomatology an unsuspected calculus cholecystitis surprised both the physician and patient. We found that in 4.1 per cent the most distinct manifestations were those of a dyspeptic syndrome without pain or jaundice and in this category it is suggested that surgical exploration be directed toward the gallbladder. Among the patients with obstructive jaundice there were about 2 per cent without a previous history of colic and the indication of surgery was based

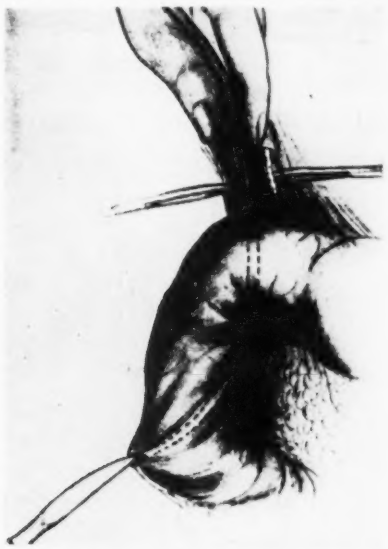


Fig. 3. Amplifying the duodenotomy (*Delfor del Valle*).

upon the doubtful diagnosis of obstructive jaundice. I have successfully operated upon only two cases of biliary ileus caused by gigantic gallstones which migrated into the ileum through a cholecystoduodenal fistula.

TABLE III

PATIENTS WITH ONE OR MORE OPERATIONS

	Cases	Percentage
Patients with only one operation	341	93.6
Patients with more than one operation	23	6.3
Total	364	99.9

In 6.3 per cent with multiple operations the author performed more than half while the balance were seen in consultation. The indications for subsequent surgical intervention were cholecystostomy, residual gallstones, odditis, and chronic pancreatitis.

TABLE IV

TYPES OF OPERATIONS PERFORMED

	Cases	Percentage
A. Cholecystectomy	211	56.7
B. Cholecystectomy and choledochostomy	65	17.4
C. Choledochostomy	25	6.7
D. Exploratory laparotomy	23	6.1
E. Cholecystostomy	18	4.8
F. Plastic, reconstruction of channels ampulla of Vater	7	1.8
G. Anastomosis of the common bile duct to the duodenum or jejunum	4	1.0
H. Cholecystostomy and choledochostomy	3	0.8
I. Abscess of gallbladder origin	1	0.2
J. Section of adhesions	14	3.7
Total of Operations	372	

Cholecystectomy alone or together with choledochostomy was carried out in 74.1 per cent of the cases. Cholecystostomy was performed in very few cases

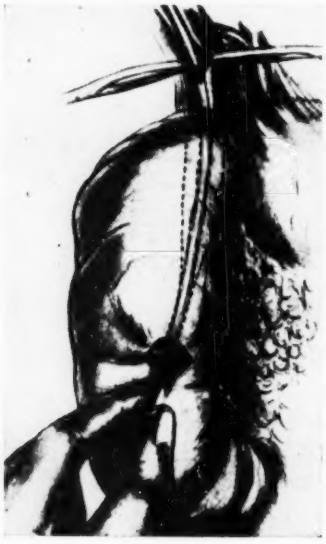


Fig. 4

and only when manifest lesions of the pancreas, cysts and advanced cirrhosis were observed. In 17.4 per cent where cholecystectomy and choledochostomy were performed, the presence of stones, a dilated common duct and pancreatic lesions were the decisive indications. The treatment of channel stenosis was a direct approach except in a few cases where the dilated portion was anastomosed to the duodenum or jejunum. In 27.4 per cent surgery of the channel included procedures from simple cholecystectomy, sphincterotomy to plastic and reconstructive surgery.

Residual Gallstones

The usual manifestations are those of a painful syndrome characterized by biliary colics, intermittent or persistent jaundice and associated infection. Early surgical intervention is necessary to avoid the complications of chronic hepatitis or retention cirrhosis. In the majority of cases medical treatment has proven quite useless. The primary operation determines the prognosis, for it is at that time that the ducts must be carefully examined even when no antecedent history of jaundice is elicited. Gallstones which are not embedded may permit a free flow of bile and may fail to promote biliary obstruction. Common duct exploration is required in the presence of dilatation, numerous small stones in the gallbladder and in particular, a previous history of jaundice. When palpation of the common duct is inadequate, then exploration, soundings, cholangiography and the use of stone detectors, which amplifies the sound of a metallic catheter, may all be

Fig. 5

necessary to reassure the surgeon. When doubt persists, in spite of all the aforementioned methods, I recommend exploration of the duodenum and the ampulla of Vater. The diagnosis of residual calculae in the common duct calls for surgical intervention at which time a Kerh sound is inserted after removal of the stones. I avoid the passage of the duodenal branch of the "T"-tube into the duodenum to deflect the regurgitation of duodenal content into the ducts. This permits a more satisfactory demonstration when postoperative cholangiography is instituted.

The presence of stones in the ducts in spite of careful surgical exploration may result in their descent through the superior hepatic ducts after the operation.

TABLE V

From 1945 to 1951	Cases	Percentage
Operations performed by the author	372	
Removals of residual calculi	17	4.8

Oddism and Odditis

When after cholecystectomy with careful exploration the ducts and abdominal viscera are found to be normal, then the attack of pain reminiscent of previous biliary colics will be the result of the syndrome created by the sphincter of Oddi. The crisis frequently results from dietary indiscretions, and following the administration of barbiturates, morphine, demerol or dolantine.

Delfor del Valle called hypertonicity of the sphinctor of Oddi with dyssynergia causing biliary hypertension, Oddism. It is a functional disorder which requires relatively simple management and offers a benign prognosis. The principle manifestations are epigastric pain which radiates toward the liver, back

Fig. 6

and right shoulder and is accompanied by nausea and even vomiting. This afebrile state is furthermore accompanied by a normal van den Bergh, while the urine is free from biliary elements. It is generally of short duration and is effectively treated with amyl nitrite, nitroglycerin, papaverine and atropine.

Since gastric hyperacidity provokes spasm of the sphincter of Oddi, antacid medication combined with an appropriate diet will favorably modify the condition of the patient. We have used with advantage periodic duodenal soundings; we furthermore advise a Boyden breakfast twice weekly, physical and mental

rest with a sojourn at mineral spas, thus completing the full management of Oddism. Where repeated episodes occur, the problem becomes chronic in which a normal level is never reached during remissions. There is more or less a pronounced jaundice which may disappear, while during intermissions a positive van den Bergh and choluria may supervene. Fever is not infrequent during a crisis.

The pain is preferably localized to the epigastrium and on palpation reaches a maximum tenderness in Chauffard's zone. The right costovertebral zone is likewise frequently found painful. Thus the picture of Oddism is reconstructed by the previous description. In the presence of jaundice, where the bile appears

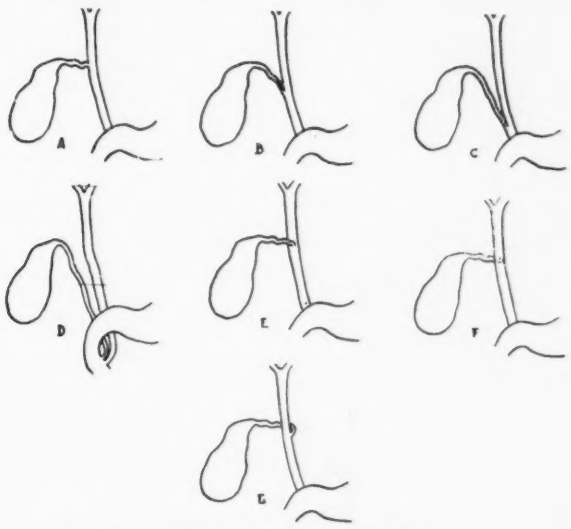


Fig. 7—A. Perpendicular approach to the common bile duct.
B. Approach in a sharp angle.
C. The cystic duct runs into the pancreatic portion of the common bile duct.
D. Outlets separated into the duodenum (very seldom).
E. In the front wall of the common bile duct.
F. In the back wall of the common bile duct.
G. In its inner edges.

infected and contains clots, there exists an associated infectious complication. The administration of antibiotics is indicated and in general, one can say that the treatment recommended for Oddism does not always give definitive results. Surgical treatment will be planned even in the absence of a positive diagnosis of the anatomical nature of the problem, particularly when continued stubborness of the manifestations described offers little hope for cure.

Before describing the treatment in detail, there follows a review of the clinical anatomy, as well as the normal and pathological physiology. The entrance of the common bile duct and the duct of Wirsung into the ampulla of Vater is shown in Figure 1.

From the anatomical point of view, the sphincter of Oddi does function. According to Testut, its fibers surround both the common bile duct and the duct of Wirsung which remained separated until their confluence permits a common union at their terminus. This anatomical arrangement is seen in Figure 2 which is taken from Delfor del Valle's "Pathology and Surgery of the Sphincter of Oddi".

The surgical approach to the papilla of Vater is best attained with a transverse incision at the level of the union of the middle and lower segments of the second portion of the duodenum. The knowledge we have of the physiology of the sphincter in relation to pharmacodynamics explains some of the manifestations in patients without a gallbladder, for this organ does serve

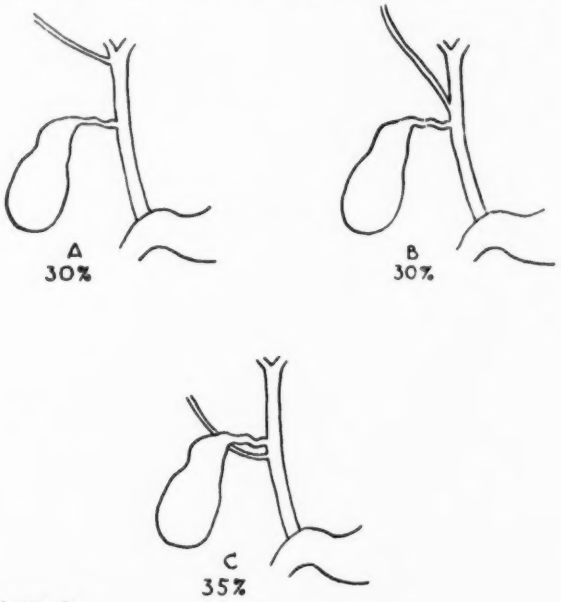


Fig. 8—Shows Flint's Findings.
A. Accessory ducts tributary to the hepatic duct.
B. Outlet very close to that of the cystic duct.
C. Ending into the common bile duct.

as a damper during the appearance of biliary hypertension. The pressure of liver bile is a little less than 35 cm. of water while that of the gallbladder is 30 cm. The sphincter opposes a resistance equivalent to 15 cm. but under pathological conditions it may rise to 75 cm. of water; then neither the liver nor the gallbladder are able to overcome this resistance. The contractions of the gallbladder are provoked by a hormone produced by the duodenal mucosa under the influence of fat which is called cholecystokinin. In addition, hyperacidity of the gastric secretions contributes to the spasm of the sphincter. Drugs such as magnesium sulfate increase bile flow through relaxation of the sphincter but are not contraindicated in the presence of gallstones since they do not influence

gallbladder contractility. Since the vagus nerve innervates the sphincter mechanism, maintaining tonus and contraction, the administration of fats, cholecystokinin, amyl nitrite, nitroglycerin, aminophyline and atropine operate therapeutically through their antivagal effect. Morphine, codeine, Pilocarpine and Demerol have a hypertonic action on the sphincter. The proper application of these agents become the basis for effective therapy.

Physiologically there are two intimately integrated mechanisms whereby the contraction of the gallbladder and relaxation of the sphincter of Oddi operate synchronously. With the emptying of the gallbladder the sphincter closes, thus facilitating the subsequent refilling of the gallbladder. Dyssynergia

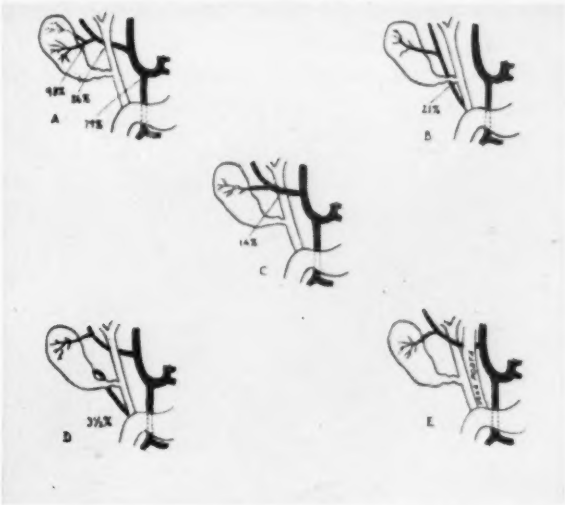


Fig. 9—A. The hepatic artery is bifurcated into the left and right branches. The latter passes behind the common bile duct. The relative frequency of the various modifications are presented by numbers in the same illustration.
B. The right hepatic artery proceeds from the superior mesentery; the left from the coeliac trunk.
C. The right hepatic artery passes in front of the common bile duct.
D. The accessory cystic that comes out of the superior mesentery.
E. The hepatic artery passes behind the portal vein (Flint).

is the inharmonious function of this mechanism which gives rise to a painful symptomatology resulting from biliary hypertension. In the absence of the gallbladder, compensatory dilatation of the ducts is insufficient to check the hypertension in the presence of a hypertonic sphincter. The unfortunate clinical picture of biliary hypertension is reproduced in those cases that contain stones in the ducts. The painful manifestations may disappear through relaxation of the sphincter brought on by the occasion of amyl nitrite.

The diagnosis of sclerosing Odditis is an urgent indication for surgical intervention to avoid infection of the small biliary ducts. Secondary operation on the duct requires the services of an experienced surgeon who, through caution and

patient effort, will overcome the many difficulties encountered. The longitudinal choledochostomy is indispensable in the exploration of the biliary ducts. Residual stones must be searched for while catheterization with a narrow sound usually permits entry through a permeable ampulla. The sound, by exerting pressure on the walls of the duodenum, facilitates exposure of the papilla.

We frequently follow the buttonhole duodenotomy technic of Delfor del Valle. The papilla may reveal different aspects: hypertrophic papillitis appearing as a nonulcerating button and easy to identify. The atrophy appears as a small orifice with converging folds. When the hypertrophic type ulcerates it may resemble a carcinoma and when it becomes fungus-like in appearance it may resemble a cancer even more so. At other times the papilla has the aspects of a strawberry.

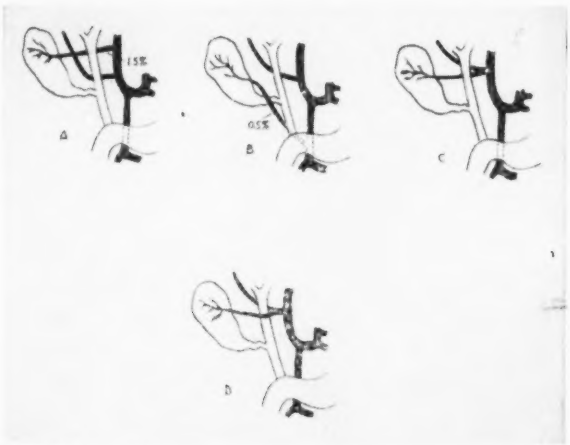


Fig. 10—Origins of the Cystic Artery.
A. Cystic artery from the left hepatic artery.
B. From the superior mesentery.
C. From the right hepatic artery near the bifurcation of the principal trunk.
D. Cystic artery from the right hepatic passing in front of the common bile duct.

After plastic surgery of the sphincter a "T"-sound with a long duodenal branch is introduced for the purpose of promoting cicatricization around the tube without forming a constriction; we permit this to remain *in situ* for two months. Following longitudinal duodenotomy suture of the duodenum is carried out transversely. The buttonhole duodenotomy is easily closed with a pocket suture. On occasion dilatation of the sphincter is performed because it is not uncommon to meet some difficulty in passing the "T"-sound. This complication is managed with a buttonhole duodenotomy followed by fixing a thread to the Benique which is passed through the duodenum and drawn out through a choledochotomy. The duodenal branch of the sound is fixed to the thread and pulled out through the duodenotomy.

Medical statistics are never precise in the analysis of Oddism. The patients at the General Hospital on my service seldom returned for late postoperative

consultation and then only when serious symptoms appeared. It is our estimate, based upon relative approximation in our private practice, that from 7 to 8 per cent of the patients demonstrate symptoms of Oddism.

Cholangiohepatitis

In some cases exposed to several surgical explorations and in spite of Kerh intubation, severe infections have been observed which we call cholangiohepatitis. This persistent infection of the small ducts extends into the hepatic parenchyma constituting an anatomical-pathological duality. In spite of satisfactory bile drainage, which is generally of a muddy and contaminated character, the jaundice persists. Frequent and repeated blood transfusions fails to affect a rather constant mycrocytic anemia. The fever daily may be intermittent or remittent and may include short afebrile periods. The general condition of the patient declines while the liver remains enlarged, soft and tender.

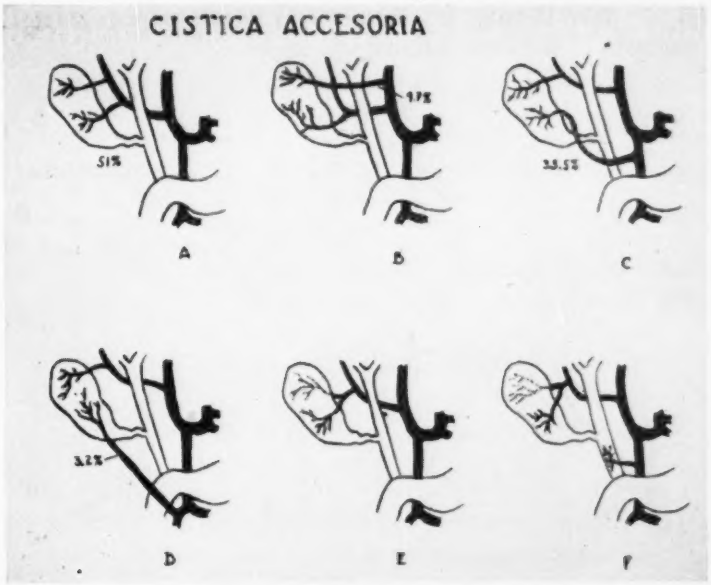


Fig. 11

The laboratory findings reveal anemia, leucocytosis and abnormal liver function tests. Though the prognosis is doubtful, cures have been obtained.

Irrigation of the ducts with warm serum supplemented with aureomycin intravenously up to 70 and 75 centigrams per day have brought cures in some cases. Chronic cholangiohepatitis due to stenosis of the biliary tract is cured by surgical intervention which permits a free flow of bile. Failure results when the infection persists in spite of canalization.

We have been impressed by the fact that occasionally the infectious process is favored by frequent regurgitations of duodenal content through the sound and it is for this reason we have maintained certain scruples against their use.

Chronic Pancreatitis

A chronic process of the pancreas has been the cause of persistent dyspeptic symptoms and pain in the postoperative course of some of my patients. Violent crises of epigastric pain with an elevated serum amylase have pointed towards the diagnosis of chronic pancreatitis with acute recurring exacerbations. At the first exploration the pancreas was hard, nodular and characteristic of diffuse pancreatitis. On other occasions, an enlarged and hard head of the pancreas was suggestive of a neoplastic process. In the main, these observations were made at the Servicio of Gastroenterology of the General Hospital and some were used by Dr. Luna for her graduate thesis. The complications of cholangiohepatitis and chronic pancreatitis were surely present in some instances at the first operation but passed unnoticed. With careful exploration during the second operation, however, diseased glands were found with definite microscopic confirmation. We systematically take biopsies of the pancreas which emphasizes the accuracy of our data because of the histopathological study.

TABLE VI

Data from 235 patients at the Servicio of Gastroenterology of the General Hospital

	Cases	Percentage
Patients with cholangiohepatitis	12	4.7
Patients with pancreatitis	33	12.9

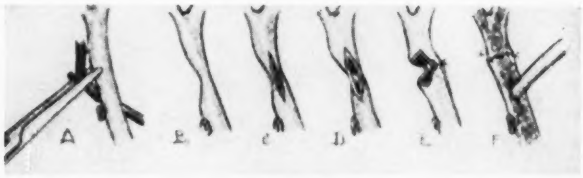


Fig. 12

Large Residual Stump of the Cystic Duct

A postoperative condition has been described, characterized by anterior abdominal pain coming on late and believed to be due to a residual cystic duct. Later on dilatation compensates for the absence of a gallbladder. It is possible that it becomes a residual inflammatory focus which gives rise to the formation of new calculae though, in reality, one or more small stones had been overlooked. It is my feeling that the long cystic stump is not the cause of postoperative symptoms, but rather the result of localized infection or neglected calculae. In one postoperative cholangiogram the long stump observed was not the cause of the postoperative manifestations, which were due to occlusion of the common duct.

Postoperative Perivesceritis

Until now I did not believe that postoperative subhepatic perivisceritis alone could cause noticeable symptoms; nevertheless, in a few patients on reoperation I could find no other explanation. According to well established technic, the surgeon must avoid the development of adhesions by removing all blood, correct peritonization, etc. The adhesions that subsequently disturb gastrointestinal motility are, undoubtedly, of great importance.

Complications of Biliary Surgery

It is exceedingly prudent to proceed with care in simple cholecystectomies because serious damage to the common duct and large blood vessels result from haste. Quite often when gallbladders are nonadherent I have demonstrated to my associates the ease with which the common duct can be accidently ligated. In spite of adequate information on the anatomy in this region the surgeon

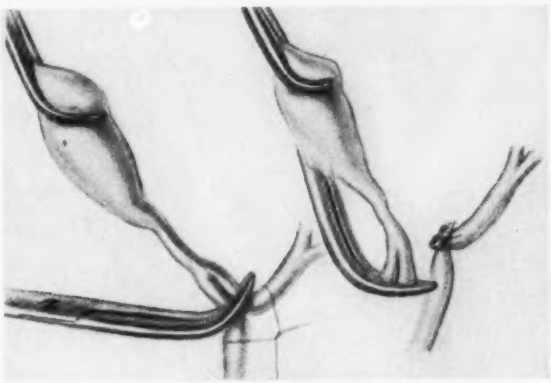


Fig. 13

should bear in mind the not unusual anomalies of the vessels and ducts which can be responsible for surgical accidents. The cystic duct can open into the common duct in several ways as illustrated in Figure 7.

Flint found accessory ducts in about 15 per cent of 200 dissections. Failure to ligate an accidently injured accessory duct may give rise to collapse and an abundant flow of bile into the abdomen. Canalizations of the infrahepatic stratum are important in the avoidance of these accidents. It is common practice to institute drainage with gauze and waterproof fabric or with a simple cigarette drain on the advice of Moyniham. There are, however, surgeons who close the abdomen with and others without drainage, but I feel no confidence in the latter procedure.

Hemorrhage is the most dreaded complication in biliary surgery and for this reason it is necessary to consider here the vascular anatomy of this region (Fig. 9).

Accessory cystic vessels, according to Flint, are found with relative frequency in about 15 per cent of the cases (Fig. 11).

Rough surgical handling and ineffective hemostasis will injure the cystic artery; loose ligatures frequently cause hemorrhage of these vessels. Attempts to control hemorrhage with hemostats in a surgical field covered with blood not uncommonly will produce injuries to the ducts that later result in stricture. It is likely too, that the same forceps may inadvertently clasp the hepatic artery. Figure 12 graphically shows this type of accident.

Lahey recommended pressure on the hepatic artery in order to control hemorrhage and thereby obtain a dry surgical field. This is performed by inserting the forefinger and thumb into the foramen of Winslow to exert pressure on the artery.

I am unaware, from the medical literature, of a similar experience observed while operating on a case of chronic cholelithiasis. At surgery we confirmed the

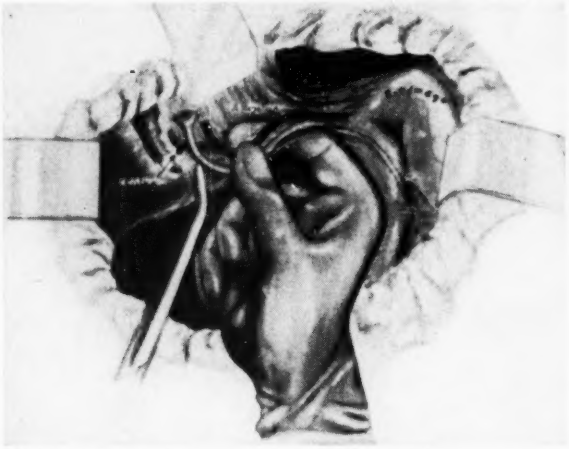


Fig. 14—Suturing of the parts of the hepatocholedochus. The narrow part has been removed.

clinical diagnosis but in addition discovered Laennec's cirrhosis without ascites. Following the cholecystectomy a massive and uncontrollable hemorrhage resulted from exaggerated traction on the round ligament. It was only through massive transfusions and tamponage with large packs that a fatality was avoided. It was necessary to leave the pack in place for 18 days in order to avoid a relapse of severe hemorrhage.

Narrowing of the Biliary Ducts

Frequent and rather constant manifestations of narrowed biliary ducts are jaundice which is aggravated by associated infection and cholangiohepatitis. Though pain is not too evident, the phenomena of infection and jaundice demands surgical intervention. At operation the possibility of residual calculae or an odditis complicated by infection of the biliary tract must be considered.

It is at the first operation that surgeons must avoid injuries to the ducts that later cause stricture. Figure 13 graphically demonstrates the danger of unwise technics.

I would like to comment on a report following a cholecystectomy in which two cystic ducts were noted. In fact, this double cystic duct was nothing less than a sectioned common bile duct seen in Figure 13. After some difficulty I was able to perform an end-to-end anastomosis. I performed a plastic operation of the narrowed duct with a longitudinal incision and a transverse suture. The "T"-sound of Kerh is indispensable provided that the branch that leaves the abdomen is pulled out directly through the suture at the point of narrowing and further down. When possible, the mucosa should be sutured to avoid future

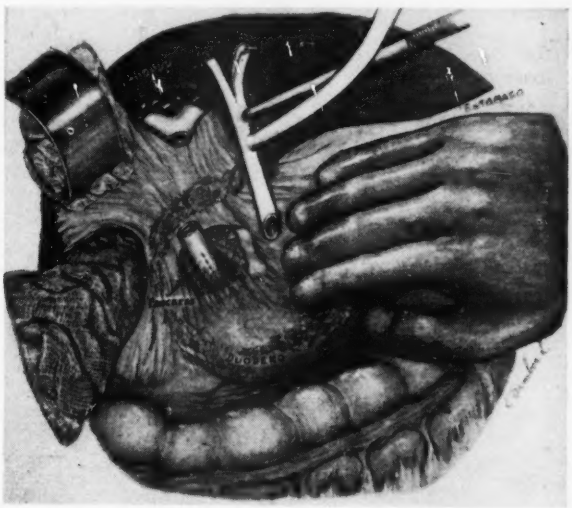


Fig. 15—Kocher's procedure to displace the duodenum and make use of more extensive portions of the retroduodenal duct.

stricture. When necessary to canalize the hepatic ducts in order to use a Lahey sound, I divide the hepatic branch, seen in Figure 14.

In urgent cases Catell performs an intrahepatic section of the ducts, a technic which I have never had the opportunity to use. I have categorically refused to perform any anastomoses between the biliary fistula and the digestive tract because I consider this operation to give precarious results. Our aim in reestablishing the function of the duct is to preserve the sphincter of Oddi whenever possible. In private practice I have anastomosed the common bile duct to the duodenum. Furthermore, in only two cases have I been obliged to leave the cannula buried in the ducts for I feared the danger of reoperation necessary to extract the sound. Both patients have had them for years and no important complications have arisen. The "T"-sound remains in the narrow duct for different lengths of time but usually more than three to four months. In several

cases I have regretted the accidental and, undoubtedly, the premature extraction of the sound through carelessness of the nurses or patient at the time of wound dressing; this may produce a recurrent stenosis.

In referring to the first illustration, seven operations performed on the biliary ducts were the result of trauma during the first surgical procedure. In one case a congenital stenosis of the common bile duct existed but in all, a permanent cure was obtained.

TABLE VII

RESULTS OF BILIARY SURGERY

Data from 364 patients operated on by the author between 1945 and 1951 who underwent 372 interventions.

	Cases	Percentage
Satisfactory results	328	90.1
Postoperative biliary complications	19	5.2
Mortality	10	2.7
Postoperative course unknown	7	1.9
	364	99.9

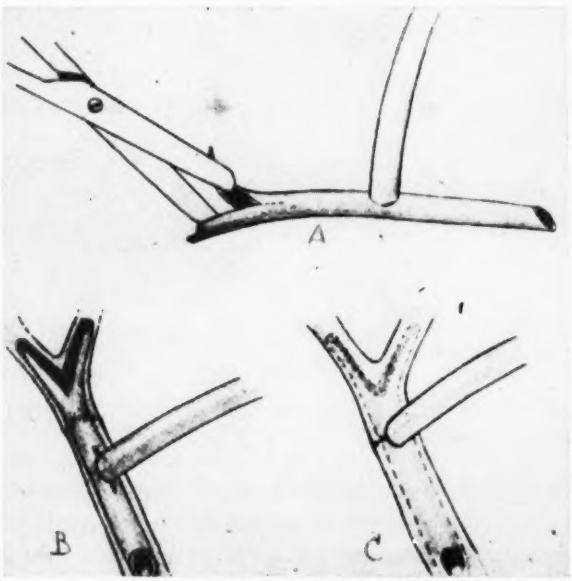


Fig. 16—Incision into the common bile duct below the suture in order to inspect it.

Satisfactory results were obtained in 90.1 per cent but this does not take into account the possibility of postoperative Oddism and associated disturbances. It does include, however, patients with secondary operations. The total mortality of 2.7 per cent is relatively low when taking into account primary and secondary operations.

TABLE VIII

OPERATIVE MORTALITY

A. Mortality consequent to the first intervention		7 in 341	2%
B. Mortality consequent to reoperation		3 in 31	9.6%
Operative mortality in the total of operations		10 in 372	2.6%
Causes of death in Group A.		Causes of death in Group B.	
Hepatic death	1	Acute hepatic insuf.	1
Hepatorenal insuf.	1	Anesthesia	1
Pulmonary embolism	1	Mesenteric thrombosis	1
Pulmonary atalectasis	1		
Myocardial infarction	1		
Unknown	1		

The analysis of the causes of death reveals a higher proportion of fatalities following the second and third operation of 9.6 per cent while the mortality following the first operation was 2 per cent. I have already emphasized the great danger in reoperation.

I hope that the audience has been satisfied with the foregoing facts. These were obtained from a statistical analysis of my own surgery, as well as from the records collected by myself and Enrique Gamboa from the Service of Gastroenterology of the General Hospital, the Gaston Melo Wards and the British-American Cowdray Hospital.

SUMMARY AND CONCLUSIONS

1. The so-called cholecystectomy syndrome was reviewed under the designation of "The Pathology and Treatment in Biliary Tract Surgery". The variety of manifestations observed in the early and late postoperative period are analyzed.

2. The pathology, prophylaxis and treatment of residual biliary calculae are generally considered.

3. The anatomy and surgical physiology is reviewed as the basis for the pathology observed in the sphincter of Oddi.

4. The survey also includes Oddism, chronic odditis, chronic cholangiohepatitis, and chronic pancreatitis. The pathology, prophylaxis and treatment of stricture of the biliary ducts concludes this study.

CHRONIC RECURRENT CHOLANGITIS CAUSED BY ANOMALY OF EXTRAHEPATIC BILE DUCTS

Case Report

SAMUEL S. GILBERT, M.D.*
Brooklyn, N. Y.
and
RUDOLPH NISSEN, M.D.†
Basel, Switzerland

Anomalies of the biliary ducts are a constant source of concern to physicians and surgeons. Numerous variations in the position and length of the cystic duct as well as the cystic artery have been described in detail[1]. Mention has frequently been made of aberrant hepatocystic ducts. Absence of the common duct has been reported less frequently[2]. Anatomic variations may lead to death or unfortunate complications if cognizance during cholecystectomy is not fully ascertained. The maneuver emphasized by Gatch[3] of tracing the biliary ducts from within after emptying the gallbladder is very important and helpful in detecting anomalies of ducts. The importance of this fact to surgeons performing in this area was again recently emphasized in a discussion by Dr. Max Thorek[4].

Cholangitis, or inflammation of the bile ducts is encountered alone as a clinical entity, or in combination with a concomitant cholecystitis. According to Walters and Snell[5] the existence of catarrhal choledochitis and cholangitis, except in combination with stones, parasites or other foreign materials in the extrahepatic bile, passages is doubtful. Classification of infections of the bile passages is listed by Walters and Snell[5]. One of their classifications is catarrhal or suppurative cholangitis secondary to ascending infections following cholecystogastrostomy or associated with cholecystoduodenal fistula. This case report describes a chronic recurrent cholangitis secondary to anomaly of extrahepatic bile ducts and postoperative choledochoduodenostomy.

Case Report

A 40-year old, white, housewife was admitted to the Brooklyn Jewish Hospital on January 23, 1949. The presenting complaint was acute pain in the right upper quadrant radiating to the back about 22 days before admission.

Past history noted intolerance to fatty foods for many years. About one year prior to this attack, she experienced an attack of acute abdominal pain, with nausea and vomiting, which required an injection of Demerol. She recalls that the pain during mild and severe episodes of distress was located in the right

*Division of Gastroenterology, Dept. of Medicine, Brooklyn Jewish Hospital.
†Formerly Attending Surgeon, Brooklyn Jewish Hospital.

upper quadrant and radiated to the right shoulder and to the scapular region. Nausea was persistent, but she vomited only on one occasion. Flatulence and constipation was the rule. Jaundice was not noticed by the patient, but was reported by her husband.

X-rays of the gallbladder taken on two previous occasions were reported as nonvisualized. Physical examination revealed a well-developed, nonobese (wt. 137), white female. Her temperature was 98.6° orally, pulse 94, blood pressure 130/85 mm. Hg. Sclerae was not icteric, skin was clear, and there was no evidence of color change. Abdomen was soft. There was slight tenderness of the upper right abdomen, but no rigidity. The liver and spleen were not palpable. Pelvic and rectal examinations were negative.

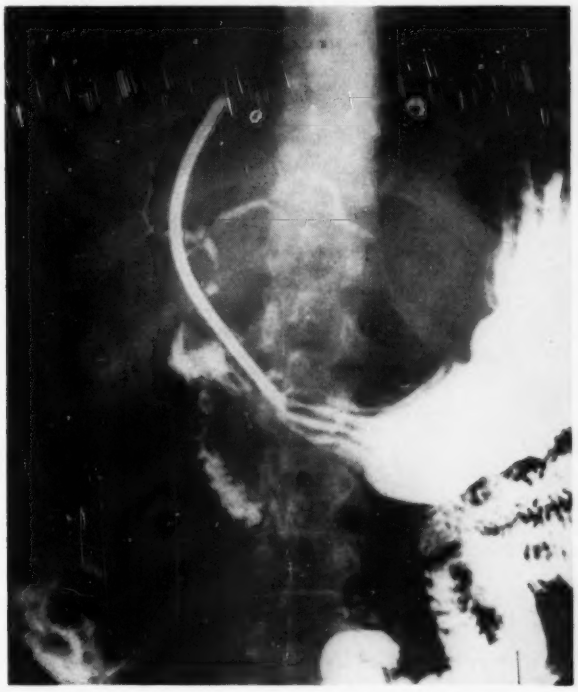


Fig. 1—Roentgenogram outlining hepatic biliary tract filled with barium. Reconstructed right hepatic duct outlined by rubber tube.

Laboratory Data:—Icterus Index was 8.5. The serum bilirubin total was 0.8 mg. per cent, the blood sugar was 70 mg. per 100 c.c. The blood urea nitrogen was 8.8 mg. per 100 c.c. Urine was within normal limits. Hemaglobin was 88 per cent. The white blood count was 9,500 with normal differential. The sedimentation rate was 12 mm. per hour. Total protein was 5.8 mg. per cent with albumen 3.9 mg. per cent and globulin 1.9 mg. per cent; serum alkaline phosphatase 5 units; cephalin flocculation and thymol turbidity reported as negative.

The provisional diagnosis was chronic cholecystitis with cholelithiasis.

Patient was operated January 24, 1949.

Operative Findings:—A small and a large calculus was found in the gallbladder. The ductal system was found to be anomalous. There seemed to be two common ducts, and an extremely short cystic duct. Opinion at surgery was, that four hepatic ducts formed two common ducts. There was a block of one common duct at the duodenal opening.

Procedure recorded was ligation of cystic artery and cystic duct. It was found that the short end of the cystic duct which was cut before it entered the gallbladder, made exit from the lower end of the gallbladder forming what was considered the second common duct. The gallbladder was only partially resected and the remainder was used to reconstruct an anastomosis with the upper

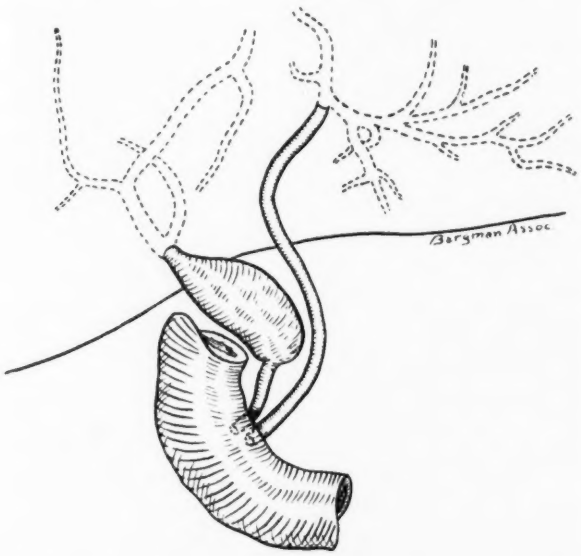


Fig. 2—Anomalous ductal system. The right hepatic duct entering gallbladder and making exit at the lower end and continuing on to the duodenal junction. The left hepatic duct communicating directly with the duodenum.

segment of cystic duct. A plastic rubber tube was inserted and retained to hold this reconstructed duct. The second so-called common duct which was not patent at the duodenal opening was subjected to a choledochoduodenostomy. Patient was discharged in good condition on February 5, 1949. During the following two years she had three attacks of acute recurrent cholangitis. Clinical picture was sudden onset with chills, fever, pain and tenderness of the right upper quadrant. The patient responded each time to a course of Aureomycin 600 mg. 24 hours, Streptomycin 1 gram daily and Demerol 75 to 100 mg. prn. On one occasion in the interval between attacks of cholangitis she noticed melena. A gastrointestinal x-ray study was made with negative findings. As commonly seen following anastomosis of bile ducts and duodenum, however, the barium

meal entered the site of the choledochoduodenostomy and flowed freely up the common duct entering the entire hepatic biliary tract which was outlined clearly as that seen in a cholangiogram. The rubber tube within the second so-called common duct was seen, but contained no barium (Fig. 1). Following the third attack of cholangitis, she was readmitted to the Brooklyn Jewish Hospital for the purpose of removing the rubber tube.

It was felt that the latter was responsible for the periodic bouts of right upper quadrant pains and abdominal distress.

A second operation was performed on February 18, 1952, two years following the original operation, at which time the ducts were found to be patent. The

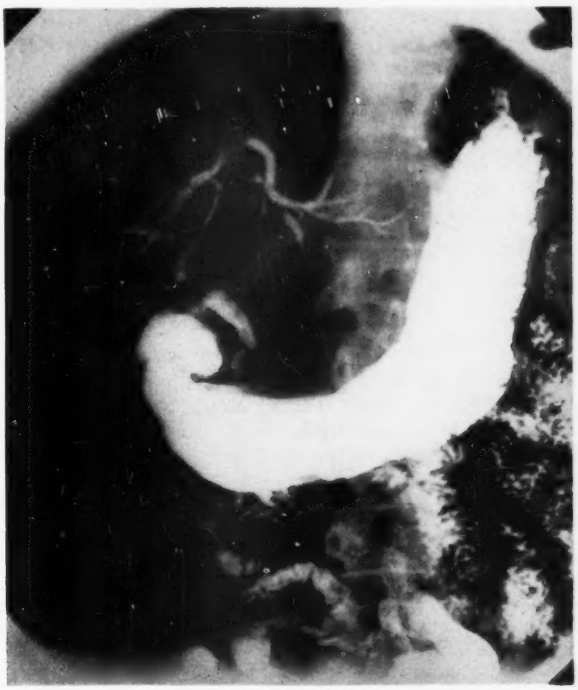


Fig. 3—Roentgenogram demonstrating barium meal ascending through the patent choledochoduodenostomy and outlining the left hepatic biliary tract.

rubber tube was removed and a rubber T-tube for drainage, was left in place and removed on the 8th postoperative day. At this operation the surgeon felt that the anomalous ductal system was best explained as two hepatic ducts. The right hepatic duct entering the gallbladder and making exit at the lower end and continuing on to the duodenal junction. The second or left hepatic duct communicated directly with the doudenal junction. The conclusion was that there was no true common duct, but in its place two hepatic ducts functioning independently (Fig. 2).

At the time of this publication 19 months following the removal of the rubber tube from the reconstructed right hepatic common duct, the patient is comparatively well.

Recent x-rays taken January 1953, reveal the barium meal entering the stomach and ascending through the patent choledochoduodenostomy and outlining the hepatic biliary tract. The right hepatic duct, which was the site of the rubber tube and reconstructed duct is not visualized (Fig. 3). Telepaque was given prior to this x-ray study in an attempt to visualize any residual gallbladder wall. There was no visualization of this structure.

Repeat of cephalin flocculation and thymol turbidity tests which were negative in 1949, were now reported as positive.

Summary

This is a case of an anomaly of the biliary ducts. It demonstrates the absence of the common duct, with two hepatic ducts replacing this structure and both functioning and entering the duodenum at separate sites. Postoperative choledochoduodenostomy contributed to an ascending recurrent cholangitis. The absence of clinical jaundice was explained on the presence of two separate ducts entering the duodenum. Subclinical hepatic damage is evident on the basis of positive cephalin flocculation and thymol turbidity tests.

REFERENCES

1. Flint, E.: Abnormalities of the right hepatic, cystic, and gastroduodenal arteries, and of bile-ducts, Brit. J. Surg. **10**:508, 1923.
2. Sachs, Allen E.: Absence of Common Bile Duct, J.A.M.A. **149**:1462 (Aug. 16), 1952.
3. Gatch, W. D.:Battersby, J. S. and Wakim, K. G.; Nature and Treatment of Cholecystitis, West Tr., S.A. (1945) **53**:380-394, 1946.
4. Thorek, Max: Surgical Errors & Safeguards. Phila., 1932, J. B. Lippincott & Co.
5. Walters, W. and Snell, A. M.: Diseases of the Gallbladder and Bile Ducts, W. B. Saunders Co. 1940.

BENTYL HYDROCHLORIDE EFFECT ON INTRAGASTRIC TEMPERATURE–NORMAL SUBJECTS*

H. B. BENJAMIN, M.D.

MARVIN WAGNER, M.D.

and

WALTER ZEIT, Ph.D.

Milwaukee, Wisc.

This paper deals with the intragastric temperatures in normal subjects under the influence of Bentyl hydrochloride† (B-diethylaminocarbethoxybicyclohexyl hydrochloride). Its effect on the regional temperature and on gastric secretion was noted.

Although records of the existence of gastric ulcer and its complications of hemorrhage and perforation were available as early as the tenth century, Cruveihlier's (1829-1835) thorough and accurate description of gastric ulcer was the first formally presented in the medical literature[17]. The works of von Jaksch and Rokitansky did much to extend our knowledge of this subject. They believed that simple gastric ulcer occurred because of the destruction of gastric epithelium, disturbed local circulation, and hyperacidity. More recently, the nervous element was suggested by Wolf and Wolff[53] as an additional causative factor, and temporary remissions have been reported as a result of psychoanalysis[2]. Regardless of the type of therapy applied, however, recurrences are common.

The numerous approaches to the therapy of gastric ulcer indicates that the etiology of the disease has not yet been clearly defined. Various aspects of and modes of approach to the problem are contained in the voluminous literature on the subject but a physioanatomical basis has heretofore never been described.

It is an accepted fact that the development of ulcers is the result of localized necrosis. The causative factors which precipitate or allow the pathological process of ulceration to progress have been the subject of much conjecture[4,5,12,15,16].

Poor dietary habits, disturbed hormonal influences, psychic stress, alcohol or excessive use of tobacco, infection, allergy, gastric endartery spasm or thrombosis, vagal overactivity and disturbed enzyme balance have been considered by some investigators[1,3,7,8,9,18,19,22,23]. Impairment or failure of the blood supply undoubtedly leads to localized necrosis and the neurovascular mechanism involved has been reported previously by Benjamin[8,10,11].

---

*Read before the Central Regional Meeting of the National Gastroenterological Association, Milwaukee, Wisc., 28 March 1954.

†Bentyl hydrochloride is the trade-mark of the Wm. S. Merrell Company, Cincinnati, Ohio, for its brand of dicyclomine hydrochloride.

From the Department of Anatomy, Marquette University School of Medicine.

Anatomically, the arteries of the stomach arise from two large arterial arcuates along the lesser and greater curvatures. From these arcuates, they are distributed to the ventral and dorsal walls by means of the loose areolar vascular highway situated between the muscularis and the mucous membrane. It was noted in the study that the left gastric artery arborized primarily about the cardiac-fundic area but not in the pyloric region, and that the right gastric artery, just beyond its derivation from the hepatic artery, showed a vascular pattern in the pyloric region and not in the remainder of the stomach.

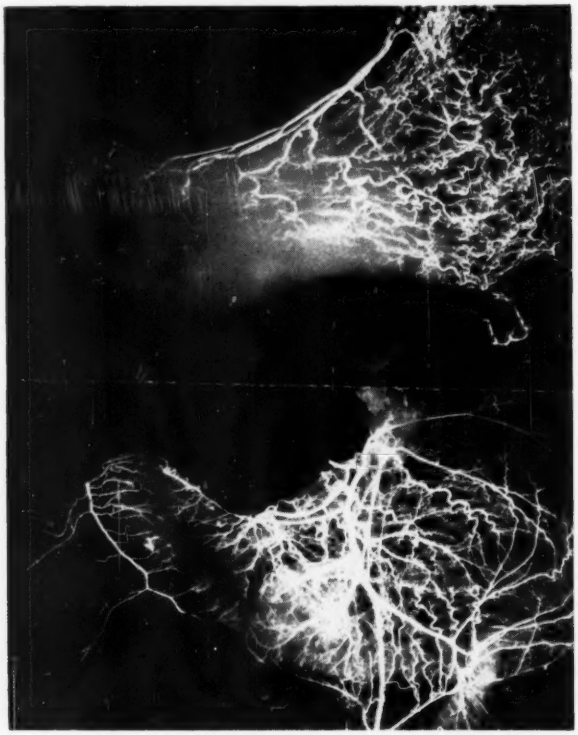


Fig. 1—The right vagus was cut and the distal stump was stimulated. The vessels were then injected. Note the absence of the vascular pattern in the pyloric region.

The effect of vagal stimulation of the regional vascular pattern was then determined in anesthetized dogs. The right vagus nerve was cut below the cardiac plexus in order to avoid direct vagal action on the heart. Stimulation of the distal stump resulted in an incomplete filling with contrast material of the vascular bed in the pyloric region of the stomach and an extensive filling of the arterial network in the cardiac-fundic area (Fig. 1). Similarly, with stimulation of the left vagus nerve subdiaphragmatically, the vascular bed was ill-defined in the cardiac-fundic area while the vessels in the pyloric and duodenal areas were well filled with the contrast material (Fig 2). It was also interesting to note that before the right vagus stump was stimulated the stom-

achs contained about 30 c.c. of viscid colorless fluid, whereas after stimulation, the gastric fluid content was considerably increased, highly acid, strongly peptic and watery. Benjamin[7] carried this study further in his consideration of this neurovascular mechanism and its relationship to the gastric mucosa[20,21,24,25,29,30]. In the unstimulated stomach the pyloric mucous membrane contained a tremendous network of interanastomosing blood vessels that extend to the limits of the mucous membrane (Fig. 3). In the stimulated stomach few of these

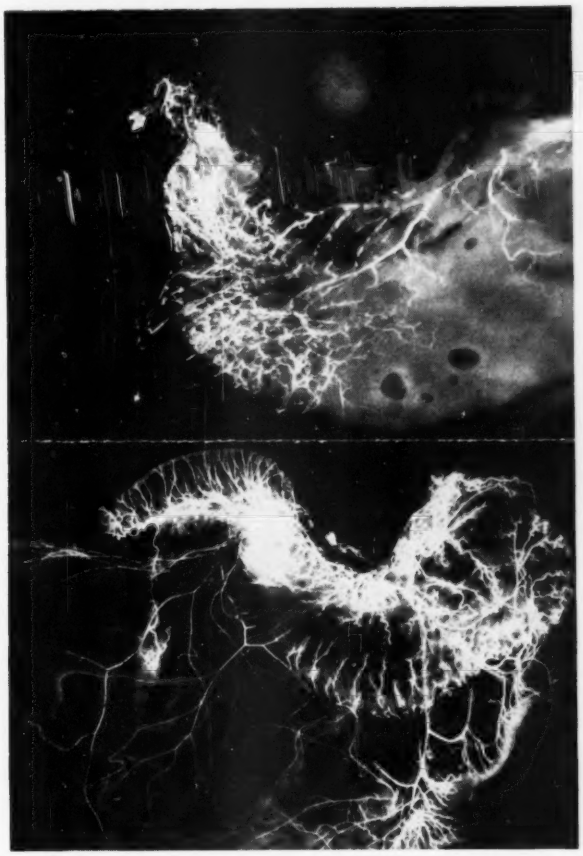


Fig. 2—The left vagus was cut. The distal stump was stimulated and the vessels were then injected. Note the plethora of vessels in the pyloric region and the absence of vessels in the cardiac-fundic areas. Numerous fenestra are apparent in the latter areas.

fine vessels were apparent, whereas the large vessels arising from the loose areolar vascular highway stand out sharply and appear stuffed with the contrast media. The main vessels showed numerous blood vessel ladders. It was apparent that stimulation of the right vagus kept the mucous membrane in a state of anemia and because of this, altered the directional flow of the blood (Figs. 4 and 5).

The results of this study of the normal and stimulated stomach support the theory that the origin of ulcers in the stomach to a great extent is dependent upon a neurovascular imbalance[36,38,39]. As Ivy, Grossman and Bachrach stated in their treatises on peptic ulcers, "The best way to account for the circumscribed nature of the acute ulcer caused by acid or any other ulcerogenic agent is by assuming a local disturbance of the blood"[26,27,28].

In direct relation to this neurovascular mechanism, we have noted variations in intragastric temperatures. As stated in a previous communication, the temperature of an area of the body or an organ must be looked upon as being variable[3,6,10,11]. Regional temperature is dependent upon rate of blood flow, oxygen consumption, and heat gained or lost through outside influences. The emotional state of the individual has a direct bearing on regional temperature, as the local vascular response (vasoconstriction or vasodilation) is regulated by

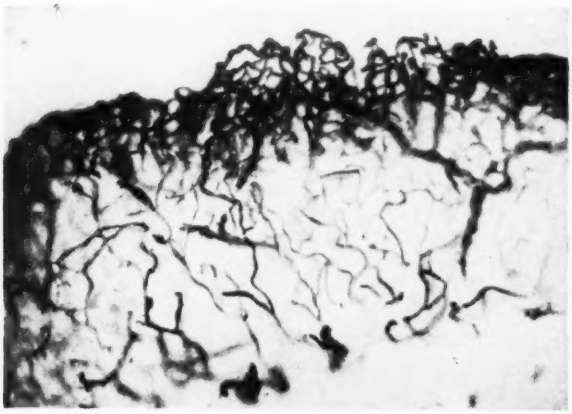


Fig. 3—Is a photograph of the injected vessels in the mucosa of a normal stomach. Note the numerous vessels. The blood vessels extend to the extreme boundary of the venous membrane.

nervous impulses. Emotional states are also reflected in the vascular "blushing" or "blanching" of the gastrointestinal mucosa.

Tissue respiration is directly dependent upon blood flow[46,48,49,50]. In the light of rational and conservative body response the blood requirements of an organ or tissue at rest would not be equal to the requirements of the same structure during different degrees of activity. As we have demonstrated previously[7], a shunting or bypassing should be a normal physiological phenomenon with blood being diverted to the tissue or organ with the greatest need[47,51,52]. Kottke[31] noted that hypoxia decreased the ability of men to control body temperature and, with the inhibition of shivering, favored greater heat dissipation. Hypoxia causes hypoxemia and this, in turn, leads to anoxia of a greater or less degree in areas activated regionally because of body demands[32-35,37].

The composite individual is the best example of division of labor. Each organ or tissue is dependent upon each other tissue or organ for its very existence. In the normally functioning individual, numerous episodes of autotransfusions from tissue to tissue and organ to organ take place for the purpose of tissue metabolism[42-45]. E. Hohwii Christensen[13,14] showed that there was a considerable reduction of the blood flow in the skin at the beginning of exertion. In a few minutes the initial constriction of the skin vessels gives way to vasodilation. This serves as a heat regulation mechanism. His experiments show that at the start of heavy work there is an instantaneous and vigorous reduction in the finger blood flow. This lasts several minutes. It is a regulatory phenomenon for the adjustment of the circulation at the transition point from rest to work. He felt that the response was too sudden to be motivated by a hormonal adrenal secretion.

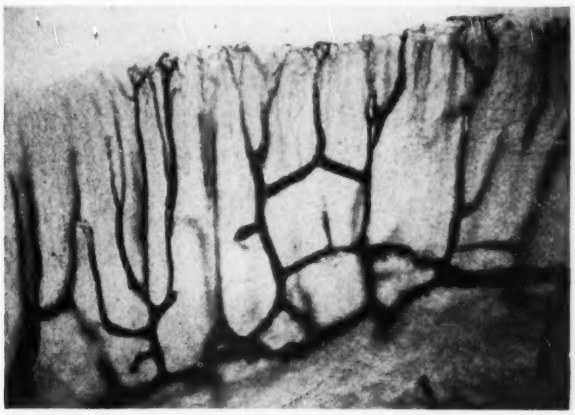


Fig. 4—Note the stepladder arrangement of the major vessels in the mucosa of the ulcerated stomach. This photograph demonstrates the short route the blood takes entering and leaving the mucosa and therefore the absence of the numerous vessels normally present in this layer.

Since a change in temperature follows upon any regional change in blood flow, and therefore oxygen exchange, there should be a concomitant temperature change if the oxygen is not used to elaborate a definite product. When the blood volume and rate of flow to an area are increased, the temperature should follow suit, and, conversely, if the blood volume and rate of flow are reduced the temperature in the area should fall.

In the experiments performed on our normal subjects with the intragastric thermopile, approximately one-half hour following the patient's desire for food the temperature took a sharp dip to a lower level, and shortly after hunger contractions ceased, became elevated to its previous or near previous plateau (Fig. 6). It was felt that this reaction took place because the muscular contractions of the stomach arrested the blood flow and led to a dearth of oxygen and a reduction of regional metabolism. In our ulcer cases, the temperature

variations were apparently related to the degree of activity of the ulcer. Activity of an ulcer is as a rule associated with hypersecretion, and as activity is always attended by increased blood flow and oxygen consumption, the temperature must of necessity follow suit. It was also interesting to note that the oral temperature was always two to three degrees below the intragastric temperatures. This contention is based on the work as elicited above, where it was demonstrated that in the stimulated stomach the mucous membrane is kept in a state of anemia because of the by-passing mechanisms of the vascular bed.

In view of the findings above, we were interested to note the effect of an antispasmodic drug on the intragastric temperature[40,41]. Our choice of drug was Bentyl hydrochloride. Chemically speaking, this compound is diethylaminocarbethoxybicyclohexyl hydrochloride. Bentyl resembles atropine and its homologues in that Bentyl has a selective relaxing effect upon smooth musculature,

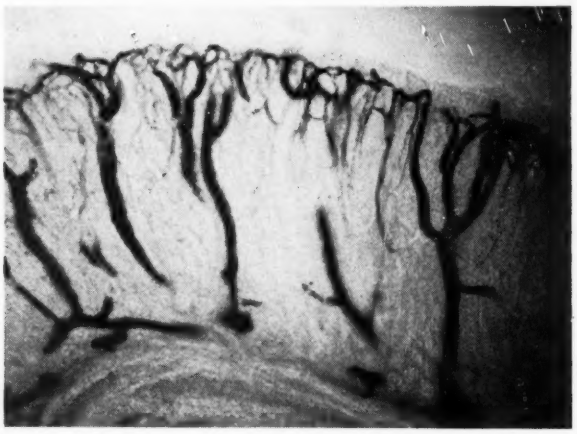


Fig. 5—Is a photograph of the injected vessels in the gastric mucosa of an ulcerated stomach. Note the lack of small tufted vessels normally present. The fractured pattern in the muscularis layer is the usual finding when an ulcer is present .

particularly of the gastrointestinal tract. The mechanism of this action is due to the parasympathetic depressant properties of the drug. It offers, however, the advantage of only minimal side-effects, such as cycloplegia, xerostomia and mydriasis. In our findings we have noted that Bentyl, because of its antispasmodic action, causes a plateau effect on our temperature curves (Fig. 7), obviating a neurovascular response, or gastric contractions and consequent gastric mucosal anemia. Undoubtedly, due to this antispasmodic effect, the stomach has a delayed emptying time, for in our subjects we have found, on gastric aspiration, food particles which were ingested four to six hours previously (Fig. 8). The gastric acidity showed no significant changes in an evaluation of over 100 cases.

Material and Methods:—One hundred freshman medical students from Marquette University who volunteered to be subjects were studied. After inter-

views to determine the fact that they suffered from no chronic disease, that they did not have ulcers, and that they were not ill at the time, we obtained a fasting gastric specimen and took the intragastric temperature by passing the thermopile into the stomach. As a rule, the temperatures were taken during the hunger phase of the individual. For example, the routine experiment began about ten o'clock and ran through the noon hour at which time the students were accustomed to having their lunch. The temperature was recorded until the subject no longer felt hungry. All the subjects showed a high take-off and with hunger contractions the temperatures fell. When the hunger pangs disappeared the temperatures began to climb.

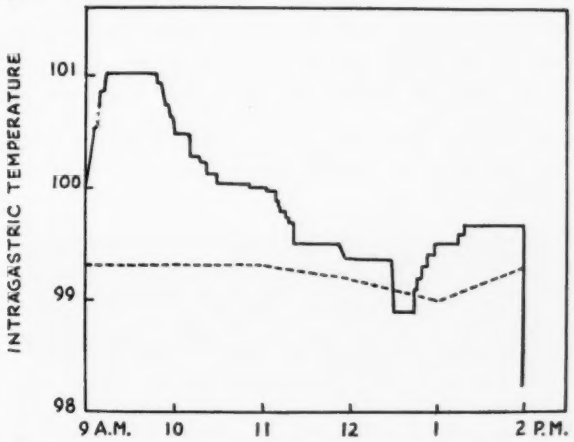


Fig. 6—The solid line shows the intragastric temperature prior to, during, and after the hunger contractions subsided. The interrupted line shows the intragastric temperature after the ingestion of Bentyl hydrochloride. The intragastric temperature after medication leveled off and did not show the fall during the hunger phase.

The student subjects were then given Bentyl hydrochloride, one capsule (10 mg.) to be taken before each meal, and one before bedtime for a period of two weeks. At the end of that time the student returned for a fasting gastric sample and for an intragastric temperature. In all our subjects, as a rule, we found that the Bentyl hydrochloride had no effect on either the free, combined, or total hydrochloric acid; but that the temperature, as recorded by the continuous recording mechanism, remained in a static phase, that is, if the patient had a high intragastric temperature, the temperature recorded showed constant during his supposed hunger period and remained elevated, showing no tendency to approach lower levels, as it did in the subjects who had received no medication. On the other hand, in those subjects where the temperature was ordinarily low intragastrically, the temperature remained low and constant without the dip during the hunger period as was found in the same subjects prior to medication.

A sample group from the one hundred freshman volunteers were intubated with an intragastric balloon to record intragastric contractions prior to and

after medication. In the subjects who had intragastric contractions taken prior to the medication, all of them showed the pronounced gastric contractions during the hunger period but after the use of Bentyl hydrochloride the spike of the gastric contraction became much lower and the intervals between the gastric contractions became wider, showing that the emptying time was therefore prolonged allowing for a smoother flow of blood through the stomach as well as through the mucosa of the stomach, and showing no states of hunger contraction anemia of the mucous membrane. These contractions were recorded by a Phipps and Bird kymograph.

With the above findings it would be apropos to review quickly the fundamental concepts of the nervous and chemical control as it applies to the autonomic nervous system, and then show its application to our work. There is evidence for the existence of levels of integrated function in the antonomic

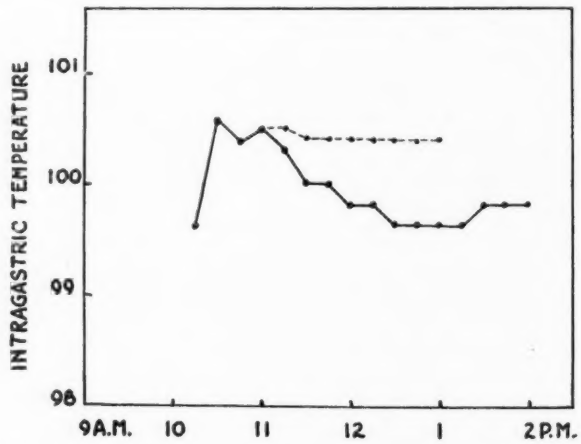


Fig. 7—The solid line shows the intragastric temperature prior to Bentyl hydrochloride. The interrupted line shows the intragastric temperature after two weeks of Bentyl hydrochloride taken by mouth. The postmedication curve does not show the drop in temperature demonstrated by the curve plotted prior to medication.

nervous system as in the somatic branchial system. The lowest degree of integration of visceral function is carried out at the peripheral level. Activity consists of axon reflexes and receptor effect or responses, as demonstrated by the myenteric reflex in the alimentary tract.

Simple reflex responses are integrated in the spinal cord. In the thoracic cord there are autonomic pylomotor, sweat and vasomotor centers whereas in the lumbar and sacral region there are the centers that control the pelvic organs. The response to temperature demonstrates integration at this level. If one hand is immersed in ice water, the other will show reflex vasoconstriction. More complex visceral responses are integrated in the medulla, where cardiac, vasomotor, respiratory and other centers are located. Swallowing and coughing are examples of integration of visceral and somatic responses at this level.

Complete integrations are carried out in the hypothalamus, such as the regulation of body temperature, and the regulation of water balance. It integrates somatic and autonomic reactions. Finally, for completion, there is the cortical level, where there is some degree of voluntary integration, as demonstrated by modifications of breathing and evacuation of the bladder.

The motor fibers are either cholinergic or adrenergic, depending upon which of two chemical mediators is released at the site of axonal termination. Cholinergic fibers liberate acetylcholine and include all visceral motor preganglionic fibers; parasympathetic preganglionic fibers, such as the vagus, as well as the sympathetic postganglionic fibers are cholinergic. Thus, all peripheral motor nerve fibers originating in the central nervous system are cholinergic. Adrenergic fibers liberate sympathin (adrenalin [epinephrine] or noradrenalin [arterenol] or both). For the most part, sympathetic postganglionic fibers are adrenergic. The exceptions are the fibers to the sweat glands, some vasodilator fibers and some fibers to the smooth muscle of the uterus, all of which are cholinergic.

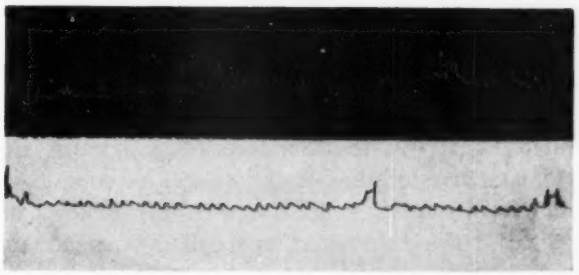


Fig. 8—Shows the gastric contractions in a normal stomach prior to and after the ingestion of Bentyl hydrochloride.

In any approach to the treatment involving the autonomic nervous system, there are three common sites of action: 1. Ganglia (acetylcholine) stimulant; 2. Effectors of the sympathetic division (sympathine-stimulant); 3. Effectors of the parasympathetic division (acetylcholine-stimulant). There are newer drugs which act both at the ganglia and at the termination of the parasympathetic fibers.

With the data we have accumulated, the following observations were made: (1) That Christensen's results in his skin experiments so closely parallel the findings in our study of the intragastric temperatures that the only logical conclusion is that the phenomena were the same. (2) The temperature changes are due to a change in the volume of blood in the vascular bed at the site of taking, and this was entirely dependent upon the status of the neurovascular mechanism, stimulated or unstimulated. (3) The level involved in this neurovascular mechanism is the integrating function of the autonomic nervous system, including the spinal cord, medulla and hypothalamus.

Summary

Tissue respiration is directly dependent upon blood flow. It is, therefore, important to think of the body architecture as physiological anatomy. In the light of rational and conservative body response, the blood requirements of an organ or tissue at rest would not be equal to the requirements of the same structure during the different degrees of activity. It becomes obvious, therefore, that shunting or by-passing should be a normal physiological phenomenon. Blood is diverted to the tissue or organ with the greatest need.

Since a change in temperature follows upon any regional change in blood flow, and therefore oxygen exchange, experiments were devised to demonstrate the relationship between the vascular pattern and intragastric temperatures. It seemed reasonable to suppose, therefore, that if the blood volume and rate of flow to an area were increased, the temperature would rise; and that, consequently, if the blood volume and rate of flow were reduced the temperature in the area would fall.

In our experiments it was noted that approximately one-half hour after the patient began to desire food, the temperature took a sharp dip to a lower level; shortly after the subject's "hunger pangs" had subsided, the temperature within the stomach returned to its previous plateau or near it.

It was postulated that this reaction took place because the muscular contractions of the stomach arrested the blood flow and led to a dearth of oxygen and a reduction of regional metabolism. This contention is based on the previously published work done at this institution on the neurovascular mechanism of the stomach. It has been demonstrated that in the stimulated stomach the mucous membrane is kept in a state of anemia because of the shunting mechanism of the vascular bed.

Bentyl hydrochloride has been administered to human subjects and its effects upon physiological mechanisms noted. Gastric motility was inhibited as demonstrated by means of kymograph studies and recordings of intragastric temperature. During the hunger phase it was found that the administration of Bentyl prevented temperature fluctuation and this was interpreted as indicating a constant flow of blood to the mucous membrane. The importance of this finding in connection with ulcer formation is discussed.

REFERENCES

1. Abercrombie, J.: Pathological and Practical Researches on Diseases of the Stomach, the Intestinal Canal, the Liver and Other Viscera of the Abdomen. Edinburgh: Waugh and Innes, 1828.
2. Alexander, F.: The influence of psychological factors upon gastrointestinal disturbances: a symposium. Psychoanalyt. Quat., **3**:501, 1934.
3. Bain, J. A., Rusch, H. P. and Kline, B. E.: Effect of temperature upon ultraviolet carcinogenesis with wave lengths 2,800-3,400. A. Cancer Res., **3**:610-612, 1943.
4. Barclay, A. E.: Microarteriography. Brit. J. Radiol., **20**:304-404, 1947.

5. Barclay, A. E. and Bently, F. H.: Preliminary note on shunting effect of trauma. Gastroenterology, **12**:177-183, 1949.
6. Bazett, H. C., Love, L., Newton, M., Eisenberg, L., Day, R. and Forster, R. II: Temperature changes in blood flowing in arteries and veins in man. J. Applied Physiol., **1**:3-19, 1948.
7. Benjamin, H. B.: The neurovascular mechanism of the stomach and duodenum. Surg, Gynec. & Obst., **92**:314-320, 1951.
8. Benjamin, H. B.: Intragastric tamponage for intractable esophageal, gastric or duodenal bleeding. Am. J. Surg., **80**:917-921, 1950.
9. Benjamin, H. B.: Double balloon double contrast studies of the stomach. Military Surg., **106**:134-138, 1950.
10. Benjamin, H. B., Wagner, Marvin and Zeit, Walter: Intragastric temperature variations in gastric ulcers. Surg., Gynec. & Obst., **97**:19-24, 1953.
11. Benjamin, H. B., Wagner, Marvin and Zeit, Walter: Intragastric temperature variations in man during hunger. Science, pp.118-160, 1953.
12. Bernard, C.: Lecons sur la chaleur animals. Paris: J. B. Bailliere, 1876.
13. Christensen, E. Hohwii, Hannisdahl and Bieger: Investigation of the circulation in the skin at the beginning of work. Acta. Physiol. Scandinav., **11-13**:162-170, 1942.
14. Christensen, E. Hohwii, Nielsen and Marius: Measurement of the blood flow in the skin at rest and during work at varied external temperatures. Acta. Physiol. Scandinav., Supp. **11-13**:171-174, 1942.
15. Code, C. F. and Varco, R. L.: Chronic histamine action. Proc Soc. Exper. Biol. & Med., **44**:475-477, 1940.
16. Comfort, M. W. and Osterberg, A. E.: External pancreatic secretion in cases of duodenal ulcer. Gastroenterology, **4**:85, 1945.
17. Cruveilhier, J.: Anatomie Pathologique. Pts. 10, 20 and 38. Paris: J. B. Balliere, 1835-1842.
18. Davis, R. E. and Ivy, A. C.: Thermal irritation in gastric cancer. Cancer **2**:138-143, 1949.
19. Decker, J.: Experimenteller beitrag sur etiologie der magengeschwure. Berline Klin. Wchnschr., **24**:369-371, 1887.
20. Deutsch, E., Spitzy, K. N. and Wohlrab, K.: Ueber die beeinflussung der magentemperatur durch verschiedene pharmaka. Arch. Int. Pharma. et Thera. **93**:400-411, (Apr.) 1953.
21. Frank, H. A. and Fine, J.: Traumatic shock—a study of the effect of oxygen on hemorrhagic shock. J. Clin. Invest., **22**:305, 1943.
22. Fox, W.: Diseases of the Stomach. ed. 3. Philadelphia, 1831.
23. Heiser, A.: Gastric Ulcer from hot and unchewed food. Med. Klin., **18**:1025-1027, 1922.
24. Hepburn, J. S., Eberhard, H. M. and Rieger, C. L.: Temperature of gastrointestinal tract; effect thereon of hot and cold foods and of physical therapeutic agents. Arch. Int. Med., **52**:603-615, 1933.
25. Ihre, B.: Human gastric secretion. Acta. Med. Scandinav., Suppl. **95**:1, 1938.
26. Ivy, A. C.: Gastric physiology in relationship to gastric cancer. J. Nat. Cancer Inst., **5**:313-337, 1945.
27. Ivy, A. C. and Farrell, J. I.: The experimental production of aclylia gastrica in the dog. Proc. Soc. Exper. Biol. & Med., **23**:752, 1926.
28. Johnson, J.: An Essay on Indigestion. Philadelphia, 1831.
29. Khanolkar, V. R. and Suryabai, B.: Cancer in relation to usages; three new types in India. Arch. Path. Clin., **40**:351-361, 1945.
30. Key, James A.: Blood vessels of a gastric ulcer. Brit. M. J., **2**:1464-1465, 1950.
31. Kottke, F. J., et al: Hypoxia and body temperature regulation. Am. J. Physiol. **49**:271, 1945.
32. Krogh, A.: The regulation of the supply of blood to the right heart. Skand. Arch. Physiol., **27**:227-248, 1912.
33. Idem.: The supply of oxygen to the tissues and the regulation of the capillary circulation. J. Physiol., **52**:457-474, 1919.
34. Idem.: The rate of diffusion of gases through animal tissue, etc. J. Physiol., **52**:391-408, 1919.
35. Krogh, A.: The diffusion of gases through the lungs of man. J. Physiol., **49**:271, 1945.

36. Lillehei, C. W., Dixon, H. L. and Wangensteen, O. H.: Relation of anemia and hemorrhagic shock in experimental ulcer production. Proc. Soc. Exper. Biol. & Med., **68**:125-128, 1948.
37. Loven, C.: The blood, its circulation and its importance in the nutrition of the body. Med. Arch. **73**:96, 1876.
38. Martin, G. J. and Wilkinson, J.: The neutralization of gastric acidity with anion exchange resins. Gastroenterology, **6**:315, 1946.
39. Mutch, N.: The silicates of magnesium. Brit. M. J., **1**:143, 1936.
40. McHardy, Gordon and Browne, D. C.: Clinical appraisal of gastrointestinal antispasmodics. Southern M. J., **45**:1139-1144, 1952.
41. Moe, G., Rennick, B. R., Capo, L. R. and Marshall, M. R.: Tetraethylammonium as an aid in the study of cardiovascular reflexes. Am. J. Physiol., **157**:158-167, 1949.
42. Necheles, H.: Diseases of the Digestive System. Philadelphia: Lea and Febiger, 2nd ed. 137, 1942.
43. Necheles, H.: The phenomenon of peptic ulcer. Am. J. Digest. Dis., **16**:237-242, 1949.
44. Neve, E. F.: One cause of cancer as illustrated by epithelioma in Hashmire. Brit. M. J., **2**:589-591, 1910.
45. Idem.: Kangri-burn cancer. Ind. M. Gaz., **76**:138-140, 1941.
46. Noer, R. J. and Derr, J. W.: Revascularization following experimental mesenteric vascular occlusion. Arch. Surg., **58**:576-589, 1945.
47. Sturgis, C. C.: Present status of pernicious anemia; experience with 600 cases over 8 years. Ann. Int. Med., **10**:283, 1936.
48. Truetta, Jose: Relations of Arterial and Venous Sides of the Cortical Intertubular Capillary Network. Oxford, England: Blackwell, 1947.
49. Truetta, Jose: Studies of the Renal Circulation. Oxford, England: Blackwell, 1947.
50. Weyrauch, H. B. and De Garis, C. F.: Vascular Patterns in the intestinal mesentery of the rat, normal and interrupted. An experimental study on collateral circulation. Am. J. Anat., **61**:343-372, 1937.
51. Wiggers, C. J., Opdyke, C. F. and Johnson, J. R.: Portal pressure gradients under experimental conditions, including hemorrhagic shock. Am. J. Physiol. **146**:192, 1946.
52. Wilhelmi, A. E. and Long, C. N. H.: Metabolic changes associated with hemorrhage. Ann. New York Acad. Sc., **49**:605, 1948.
53. Wolf, S. and Wolff, H. G.: Human Gastric Function. London: Oxford University Press, 1943.

EFFECTS OF SMOKING TOBACCO ON GASTRIC ACIDITY AND MOTILITY OF HOSPITAL CONTROLS AND PATIENTS WITH PEPTIC ULCER

F. STEIGMANN, M.D.

R. H. DOLEHIDE, M.D.

and

L. KAMINSKI, M.D.

Chicago, Ill.

The effect of smoking cigarettes on gastric acidity in patients with duodenal ulcer has been reported by several workers. Thus Gray[1] found that smoking on a fasting stomach increased both the gastric secretion and acidity. Friedrich[2] too found evidence of increase in volume of gastric juice and acidity or both following smoking. Ehrenfeld and Sturtevant[3] noted that in ulcer patients smoking increased the rate of response to the alcohol test meal. Crohn[4] also stated that tobacco, particularly in the form of cigarettes, increases gastric secretion and acidity. Hurst[5] considered smoking a factor in duodenal ulcer. Trowell[6] noted that the practice of inhaling was twice as common in duodenal ulcer patients as in normals and this was confirmed by others[7]. In addition to the direct effects of smoking on gastric secretion, there are well controlled experiments showing that smoking decreased the effectiveness of antacids in the management of ulcer patients[8]; others reported adverse effects of smoking on ulcer patients on purely clinical observations[9,10,11].

On the other hand, some workers found little or no increase or even depression of gastric acidity following smoking[12-16]. Moreover, some also felt that there was no difference between the effect of smoking of standard cigarettes and "denicotinized" cigarettes[1,3,17,18,19]. Despite the controversial data, Ivy[17] believes that statistical studies which minimize or negate smoking as a factor in aggravating ulcer symptoms or digestive complaints should not be taken too seriously.

Because of the controversial data and since most observers are of the opinion that the systemic effects resulting from the smoking of cigarettes are due to nicotine present in and absorbed from the smoke, the present study was planned to determine the effect of smoking standard and filtered cigarettes on gastric acidity and motility.

In this investigation two different types of cigarettes—one standard and one filter-type—were tested in 98 persons, 44 hospital controls and 54 peptic ulcer patients, by the following procedure. Fasting patients were intubated with a Levine tube and the gastric contents were aspirated twice at 10-minute intervals. After the second aspiration, the patients were given a cigarette to smoke and

From the Departments of Internal Medicine and Therapeutics of the Cook County Hospital and Department of Internal Medicine, College of Medicine, University of Illinois, Chicago, Ill.

the aspirations were continued every 10 minutes for another six aspirations. The gastric aspirations were tested for free and total acidity. The same procedure was continued for two mornings with the patient smoking a different type of cigarette each time.

The hospital controls included different types of ailments in a convalescent stage. Both men (20) and women (24) were tested. Most of the men were smokers, while many of the women were nonsmokers. Of the ulcer patients 38 were male and 16 female, all being smokers.

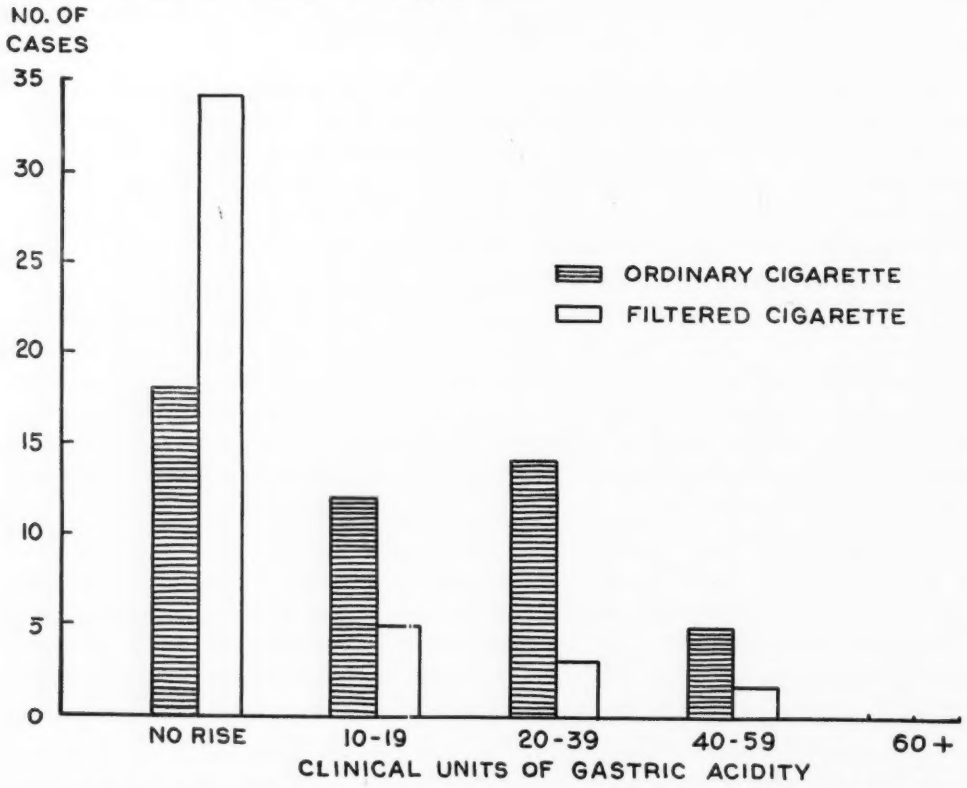


Fig. 1—Graph showing the various changes in gastric acidity following the smoking of an ordinary and filtered cigarette respectively in hospital controls.

The following procedure was used in the motility study of 16 subjects. The subjects used were male patients in the hospital in relatively good condition, recovering from various illnesses including cases of peptic ulcer—gastric or duodenal—all of them being smokers of various degrees (mild, moderate or heavy). The tracings were made after 6-12 hours of fasting. The subjects were placed in recumbent position on a comfortable bed in a very quiet room.

A Miller-Abbott tube with a balloon of about 50 c.c. capacity attached to it was passed into the stomach and placed in the antrum. The localization of the

balloon was checked with the aid of fluoroscopy and the tube was fixed to the face with adhesive tape.

The tube was connected to the water manometer, the balloon inflated with 10 c.c. of air. The recordings of the pressure in the balloon were made on a moving kymograph paper. The kymograph moved at a speed of 2 cm. per minute. The motility was recorded for a period of 2-2½ hours.

After the first control period of 30 minutes the patient was given a cigarette and the beginning and the end of the smoking period was marked on the kymo-

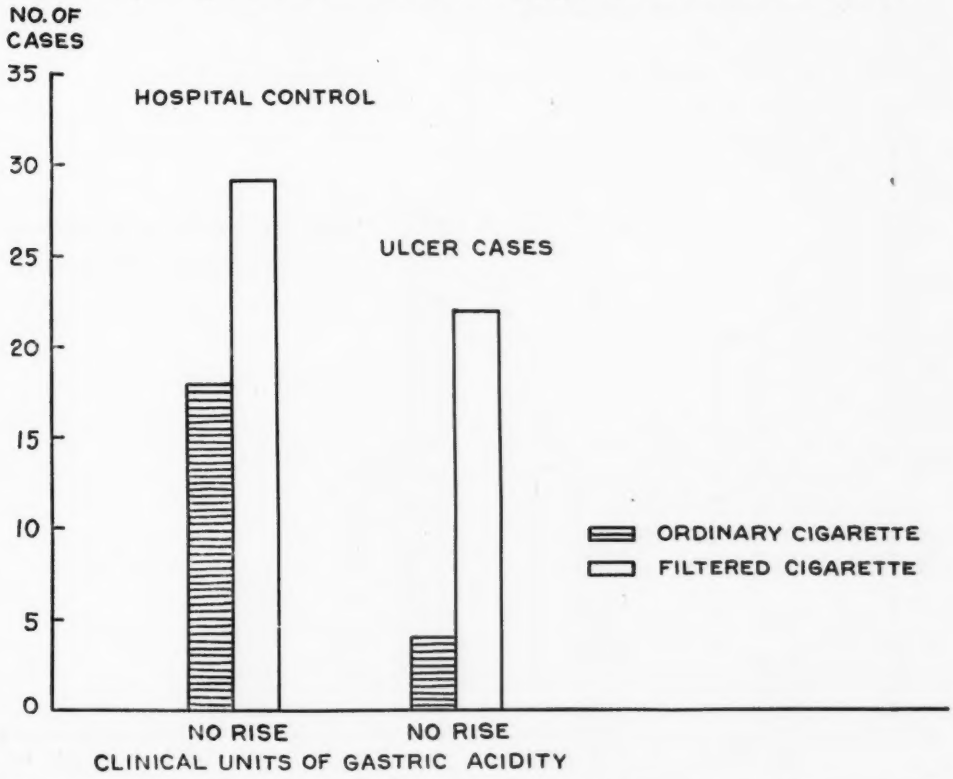


Fig. 2—Graph showing the number of cases, both hospital controls and ulcer cases, with unaffected gastric acidity after smoking an ordinary and filtered cigarette respectively.

graph paper. After this smoking period a second control period lasting 30 minutes was started. At the end of this period a second cigarette of the same kind was given and the start and end of the smoking noted. The third 30-minute long period followed the second smoking period. The subjects were closely observed and all the unusual waves due to movements or cough were marked on the moving graph paper.

The tracings of the fundus motility were made in a similar way. A balloon of about 150-200 c.c. capacity was attached to the tube which was intubated into

the stomach. Two hundred c.c. of air was injected and the tube gently withdrawn until resistance was felt indicating that the balloon reached the cardiac end of the fundus. The air was released afterwards and the tube attached to the face, connected to the manometer and the balloon was filled with 30 c.c. of air. The tracings were done as in the antral case and the periods of observation were exactly the same.

Interpretation:—Graphs were interpreted according to the height of each wave. From normal control observations in antral studies the height of approximately 0.1 to 0.5 cm. was considered as reflecting the hypoactive; from 0.6 to 1.4

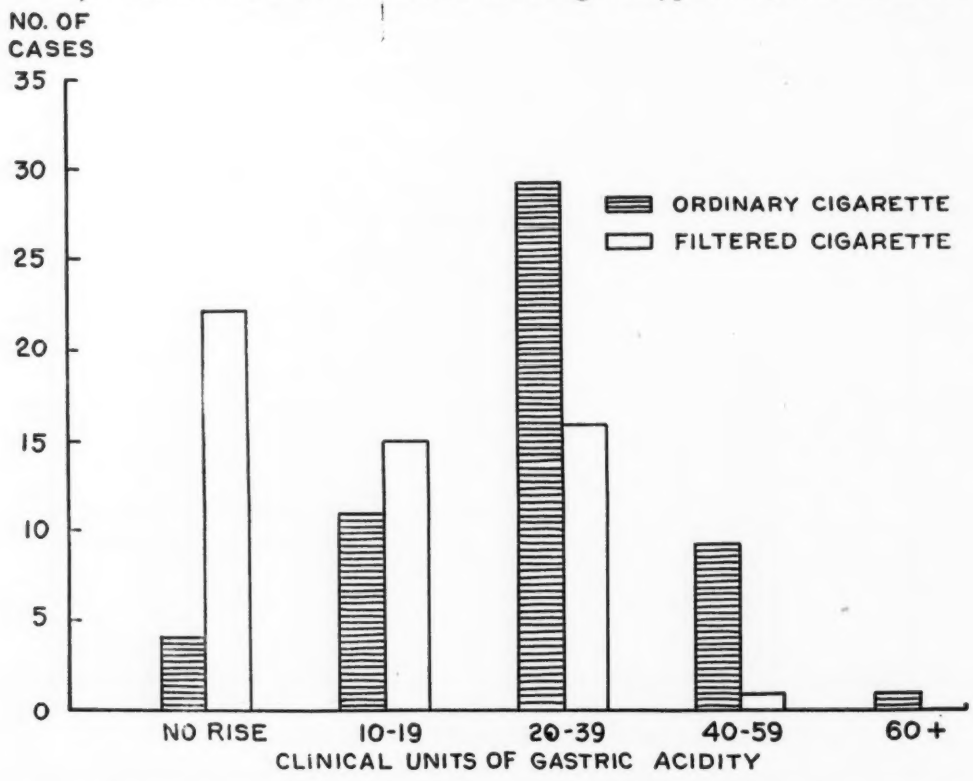


Fig. 3—Graph showing the changes in gastric acidity following the smoking of an ordinary and a filtered cigarette in patients with peptic ulcer.

cm. the average and from 1.5 cm. and over, the hyperactive motility. The width of the waves did not seem to be of any significance and hence was not considered in the interpretation.

In the motility studies in which the balloon was in the fundus, the height of the waves was different from those obtained when the balloon was in the antrum. Accordingly, in these studies waves up to 1.0 cm. were considered as hypoactive; from 1.1 to 2.5 cm. as average and over 2.5 as hyperactive motility.

Results

A. *Hospital Controls:*—Following the smoking of a standard cigarette the total acidity rose 10-19 clinical units* in 12 cases; 20-39 clinical units in 9 and 40-60 clinical units in 5 cases. In 18 cases no appreciable rise was noted (Fig. 1).

Compared to these observations are the following results found after smoking one filter-type (micronite) cigarette: a rise of 10-19 clinical units in 5 cases; of 20-39 units in 3; a rise of 40-60 in 2 cases. In 34 cases no appreciable rise was noted.

TABLE I

Changes of Gastric Motility following Cigarette Smoking in 16 Patients

			After Smoking 1st Cigarette				After Smoking 2nd Cigarette		
Site of Balloon	Type of Cigarette		Gastric Motility						
			Incr.	Decr.	Unch.		Incr.	Decr.	Unch.
Antrum	Ordinary	1st control period	0	3	7	2nd control period	2	3	5
	Filtered		1	2	7		1	0	9
Fundus	Ordinary		1	2	3		1	2	3
	Filtered		3	0	3		1	2	3

These observations indicate a definite increase in the gastric acidity following smoking of a standard cigarette in 50 per cent of the hospital controls as compared to less than 25 per cent following smoking of a filter-type cigarette.

Almost half (5 cases) of the hospital controls who reacted to the smoking had a slight rise—10-19 clinical units—following the filtered cigarette and the other half (5 cases) showed the higher acid increases (i.e. 20-39 units, and 40-60 units), while almost twice as many had no rise following the filtered cigarette (Fig. 2).

The ulcer patients behaved slightly differently. Of these 4 had no rise following a standard cigarette; 11 had 10-19; 29 had 20-39; 9 had 40-60; and 1 had a rise of 80 clinical units. Following a filtered cigarette 22 had no rise; 15 had

*One clinical unit of acid is the amount of acid per 100 c.c. of gastric juice which is neutralized by 1 c.c. of 0.1/N sodium hydroxide.

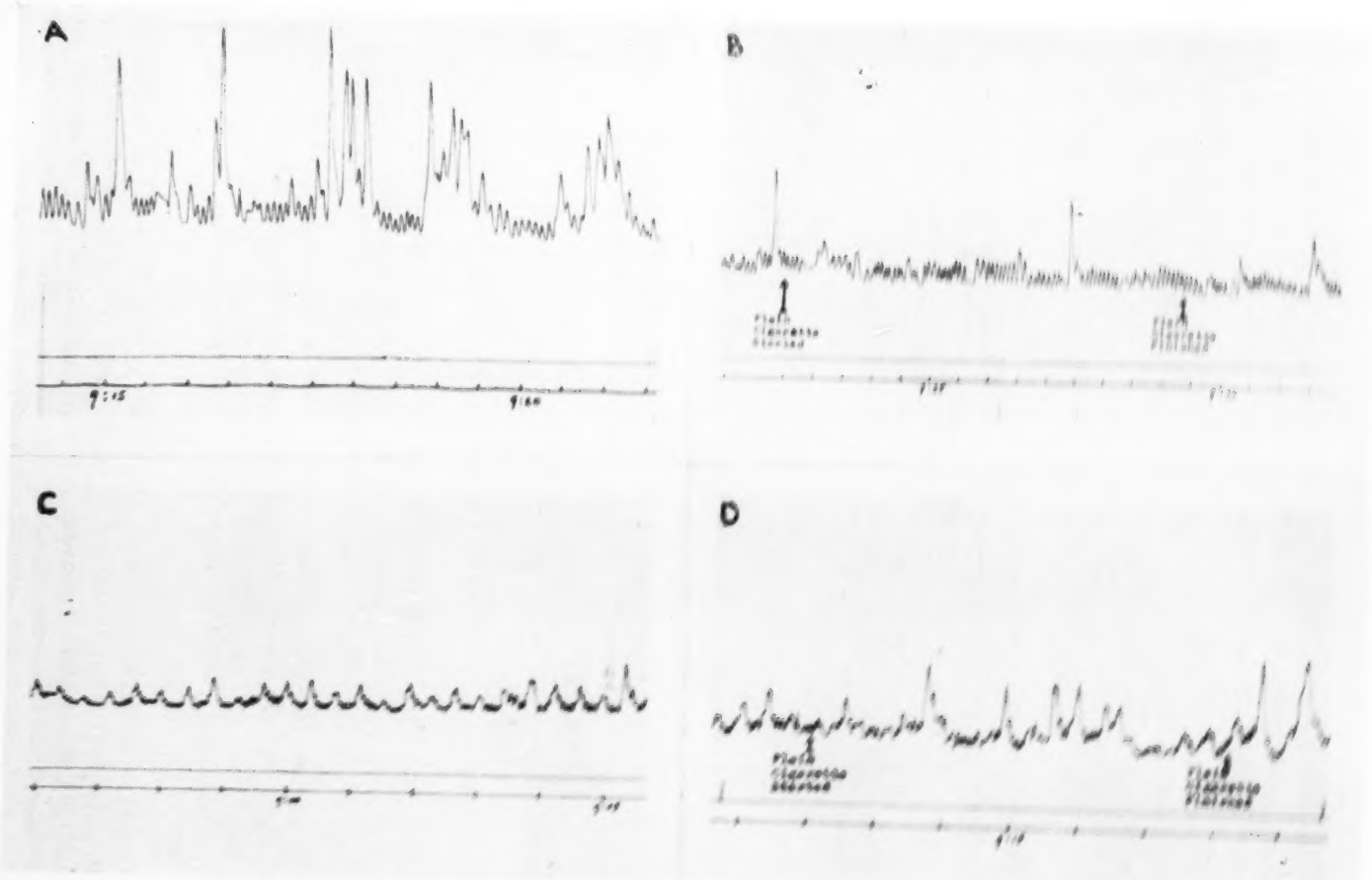


Fig. 4—Graph showing results of antral motility studies. A. Control period. B. Increased motility after smoking. C. Control period. D. Increased motility after smoking.

10-19; 16 had 20-39; 1 had 40-60 and none had over 60 clinical units (Fig. 3), while almost five times as many had no rise after the filtered cigarette (Fig. 2).

Of the 6 patients in whom complete motility studies were obtained when the balloon was in the fundus, the smoking of an ordinary cigarette caused increased motility in 1, decreased motility in 2 and unchanged motility in 3. In these patients the smoking of a filtered cigarette resulted in increase in motility in 3, unchanged motility in 3 and in none was there a decrease in motility.

After a short control period the patient was then given a second cigarette to smoke. Following this second cigarette there was, after the ordinary cigarette, increase in motility in 1, decrease in motility in 2 and unchanged motility in 3. Similar results were obtained following the smoking of the second filtered cigarette (Table I).

Ten patients were tested when the balloon was in the antrum. Following the smoking of the first ordinary cigarette there was a decrease in motility in 3 patients and unchanged motility in 7. Following the smoking of the filtered cigarette there was an increase in motility in 1, decrease in motility in 2 and unchanged motility in 7. After a short control period the patients were given a second cigarette to smoke.

After the ordinary cigarette there was an increase in motility in 2, decrease in motility in 3 and unchanged motility in 5. After the filtered cigarette there was an increase in motility in 1, decrease in motility in none and unchanged motility in 9 (Table I, Figs. 4 and 5).

Comment

While there seems to be agreement among most workers that the systemic effects resulting from the smoking of cigarettes are due to nicotine present in and absorbed from the smoke, there is some lack of agreement as to how these systemic effects manifest themselves. The latter holds especially true in regard to the gastrointestinal tract. Thus it seems that some fairly definite conclusions as to the effect of smoking have been reached in regard to the cardiovascular system[20] and to its effect on the central nervous system and striated musculature[21]. The general response of the gastrointestinal tract to cigarette smoking is, however, by no means as well defined. While in general one would expect evidence of primarily parasympathetic ganglionic stimulation with increase in gastrointestinal tone, motility and secretions, the observed effect of smoking on the gastrointestinal tract depends upon which of the autonomic ganglionic systems is more stimulated.

Our results seem to conform with the above possibilities and also demonstrate the variations that may occur. In general, however, they seem to follow a definite pattern.

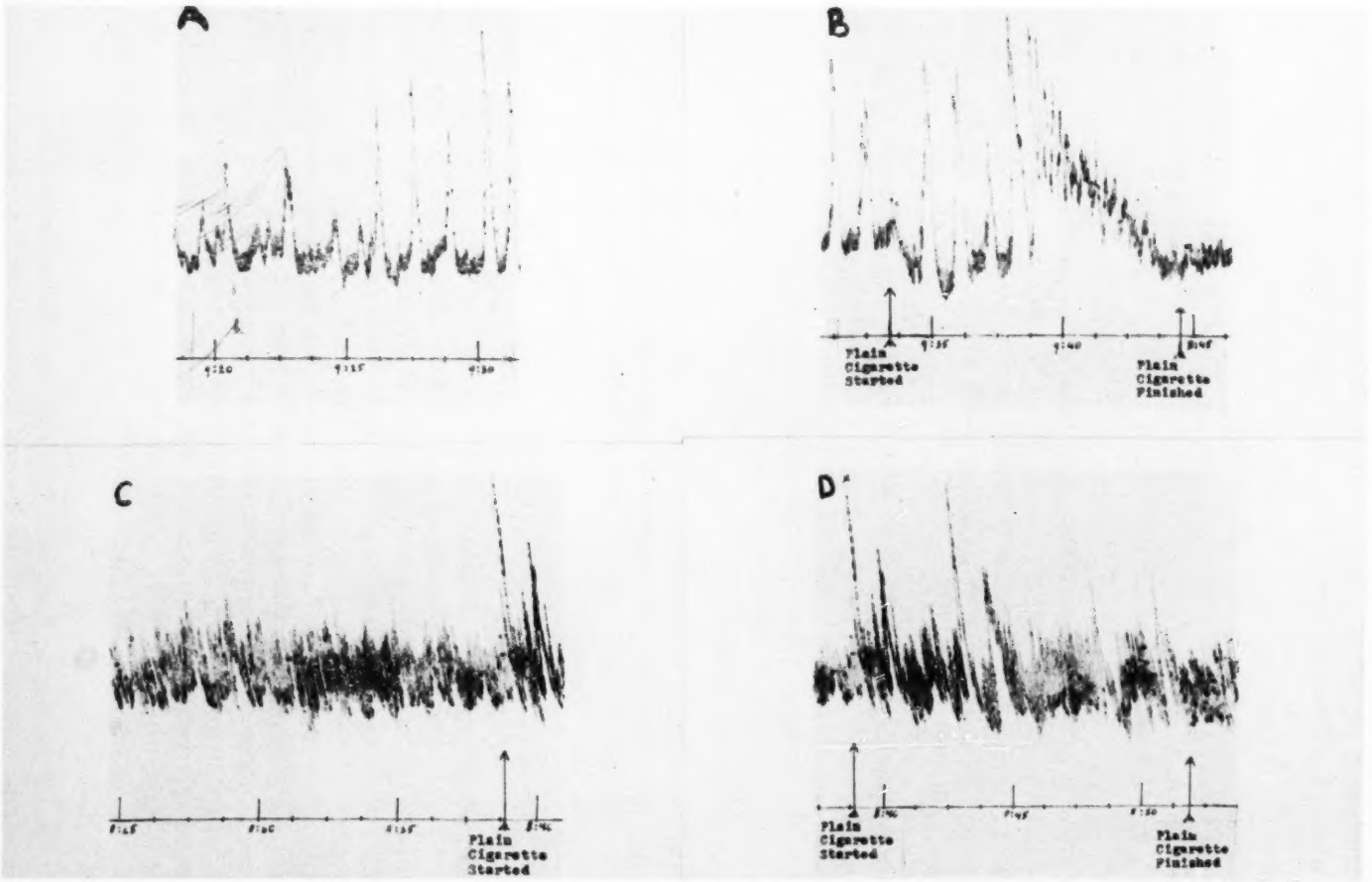


Fig. 5—Graph showing results of fundic motility study: A. Control period; B. Decreased motility after smoking; C. Control period; D. Increased motility after smoking.

Concerning the results of smoking cigarettes (nicotine effect) on the gastric acidity, there seems little doubt from the presented graphs that more than 50 per cent of the hospital controls had a rise in gastric acidity following an ordinary cigarette, as compared to less than 25 per cent after a filtered cigarette. Of the ulcer patients over 90 per cent showed a rise in gastric acidity following an ordinary cigarette as compared to about 60 per cent following a filtered cigarette. Moreover the rise in the acidity following an ordinary cigarette was in the higher ranges–40 plus clinical units–while it was in the lower ranges–10 to 40 units–following a filtered cigarette. It was also of interest that while only about twice as many hospital controls had no rise in gastric acidity following a filtered cigarette, about five times as many ulcer patients had no rise in gastric acidity following a filtered rather than an ordinary cigarette (Fig 2). While we are not prepared at this time to discuss the basis for this rise in gastric acidity, the evidence for it is clearly established.

Turning to the results of smoking on gastric motility, we find a less definite pattern, although some general course is also evident. It is especially noteworthy that the results of fundic and antral observations are somewhat at variance, which points to the difficulty in appraising the effects of smoking on the gastrointestinal tract–unless the particular part of the gastrointestinal tract is clearly defined. It appears from our observations that smoking of an ordinary cigarette will decrease antral motility about 30 per cent more often than a filtered cigarette, while after two ordinary cigarettes antral motility was 3 times as frequently decreased as after 2 filtered cigaretes. The increase in motility appeared equally divided (Table I). Fundic motility on the other hand seemed to show less decrease from smoking than the antral motility, although here too some prevalence after smoking the ordinary cigarette was noted.

The differences in the rise of acidity following smoking an ordinary cigarette and a filtered cigarette were submitted to statistical evaluation, using the "t" test* to test the significance of the differences between the means of the two samples of ulcer patients and between the two samples of hospital controls. A probability level of 0.01 was used. For the ulcer cases, a significant "t" value would be 2.678†; in our study a "t" value of 4.6 was obtained. For the hospital controls, a significant "t" value would be 2.69; in our study a "t" value of 3.1

---

* $$\text{"t"} = \frac{\overline{X}_1 - \overline{X}_2}{\sqrt{\frac{S^2_{x_1}}{N_1} + \frac{S^2_{x_2}}{N_2}}}$$

where $\overline{X}$ = the mean of the sample,

S^2_x = the variance of the mean of the sample, and

N = the size of the sample.

†Snedecor, G. W.: Statistical Methods, Ames, Iowa, Iowa State College Press, 1937.

was obtained. Therefore, the value of "t" was found to be significant in both hospital controls and ulcer patients following the smoking of an ordinary and a filtered cigarette, respectively.

These data seem to point to the fact that there are less changes in the motility of the antrum—in either direction—following the smoking of a filtered cigarette, or that there is less nicotine effect. The changes in antral motility, particularly its decrease may be of additional importance if it is considered that this may lead to poor gastric emptying with its associated sequelae. There is a possibility—which should be further investigated—that the rise in gastric acidity following smoking of an ordinary cigarette may be related to the decreased antral motility.

Conclusions

There is a definite increase in gastric acidity following smoking of a cigarette in a high percentage of both hospital controls and peptic ulcer patients.

The increase in acidity is more frequent and in the higher ranges following an ordinary than a filtered cigarette.

The antral motility is also more often decreased following the smoking of an ordinary than a filtered cigarette.

REFERENCES

1. Gray, I.: Gastric response to tobacco smoking, Am. J. Surg. **7**:489, 1929; Tobacco smoking and gastric symptoms, Ann. Int. Med., **3**:267, 1929-30; Gastric response to tobacco smoking, Tr. A. Gastroenterol. Assoc., **32**:489, 1930.
2. Friedrich, R.: Das Nicotin in der Atiologie und in der post-operativen Nachbehandlung der Ulcuskrankheit, Arch. klin. chir., **179**:9, 1934.
3. Ehrenfeld, I. and Sturtevant, M.: The effect of smoking tobacco on gastric acidity, Am. J. M. Sc., **201**:81, 1941.
4. Crohn, B. B.: Gastroduodenal ulcer; etiology, treatment and end results, New England J. Med., **218**:148, 1938.
5. Hurst, A. F. and Stewart, M. J.: "Gastric and Duodenal Ulcer," London, Oxford University Press, 1929.
6. Trowell, O. A.: The relation of tobacco smoking to the incidence of chronic duodenal ulcer, Lancet, **1**:808, 1934.
7. Jamieson, R. A., Illingsworth, C. F. W., and Scott, L. D. W.: Tobacco and ulcer dyspepsia, Brit. M. J., **2**:287, 1946.
8. Batterman, R. and Ehrenfeld, I.: Influence of tobacco smoking upon the effectiveness of antacid therapy in the management of the peptic ulcer patient, J. Clin. Invest. **27**:524, 1948.
9. Cramer, F.: Ueber den Einfluss des Nicotins, des Kaffees und des Tees auf die Verdauung, München med. Wchnschr., **54**:929, 1907.
10. Ortner, N.: Discussion of paper by Haudek, Wien. klin. wchnschr., **31**:256, 1918.
11. Rutimeyer, L.: Magenblutungen In: Kraus-Brugsch: "Spezielle Pathologie und Therapie innerer Krankheiten," **5**:1087, Berlin Urban and Schwarzenberg, 1921.
12. Schnedorf, J. G., and Ivy, A. C.: The effect of tobacco smoking on the alimentary tract, J.A.M.A., **112**:898, 1939.
13. Cramer, F.: Nikotin und Verdauung, München med. wchnschr., **72**:908, 1925.
14. Pollard, H. M. and Bachrach, W. H.: Unpublished data.

15. Barnett, C. W.: Tobacco smoking as a factor in the production of peptic ulcer and gastric neurosis, Boston M.E.S.J., **197**:457, 1927.
16. Johnson, W. H.: Tobacco smoking, J.A.M.A., **93**:665, 1929.
17. Ivy, A. C., Grossman, M. I. and Bachrach, W. H.: Peptic ulcer, p. 395, Philadelphia-Toronto, The Blakiston Company, 1950.
18. Grossman, J. and Jiano, A.: Sur les effets de l'intoxication nicotinique sur la structure fine de la cellule gastrique, Arch. mal. de l'app. digestif, **4**:369, 1910.
19. Bernay, P. and Faure, G.: Cafe decafeiné et tabac denicotinisé en dietetique gastrique, Arch. mal. de l'app. digestif, **27**:865, 1937.
20. a. Roth, G. M.: Effects of Smoking of Tobacco on the Cardiovascular System of Normal persons and patients with hypertension, J. Am. Geriatrics Soc. **2**:271, 1954.
    b. Friedell, M. T.: Effect of Cigarette Smoke on the Peripheral Vascular System, J.A.M.A. **152**:897, (July 4), 1953.
21. Haag, H. B.: Pharmacology of Tobacco: Characteristics of Cigarette Smoke, J. Am. Geriatrics Soc. **2**:274, 1954.

President's Message

The Nineteenth Annual Convention of the National Gastroenterological Association and the First Annual Convention of the American College of Gastroenterology is now history.

Its Founding Fathers, the Trustees, and its Board of Governors can well be proud of their accomplishment. The germ of an idea born in New York City, nurtured nationally and internationally, has now demonstrated its right to live and breathe.

The Washington Convention was almost everything we could ask for, and serves as an everlasting inspiration to all of us.

As your new President, I feel very humble and inadequate, but I also sense an urge and a desire on the part of the Fellows, Associate Fellows and Members to perfect what has now begun. I pledge my whole-hearted and unbiased effort to the furtherance of the objectives of the College, as laid down in the Constitution. With your help, the advice and guidance of the creators, we will build our College broad and deep—to the end that gastroenterology as a specialized field of medicine may occupy the place of dignity which it deserves.

Let us dedicate our effort this year toward consolidating and perfecting our organization beyond its present state. Let us improve and expand our membership in a healthy and orderly manner. Let us uncover and develop hidden talent among our members which we think exists, but of which we are not now aware. Let us drive through to completion the incompleted projects of our high-minded and most worthy predecessors. There is work for all. May we hear from the volunteers?

Lyon A. Ferguson

EDITORIAL

Ulcer of the Stomach and Duodenum

The months with the "R" are with us again and it is the season for recurrence of the ulcer syndrome. Why this phenomenon should occur mostly during or between September and April, is still a moot question.

We know that the increasing frequency during the past 50 years offers the strongest possible evidence that peptic ulcer is influenced by environmental factors and may well provide a key to their identity; the increase being almost entirely in respect to duodenal ulcer and principally affecting men. We also know that ulcer affects people of every social class and there is little evidence of occupational incidence.

The geographical incidence shows wide differences between negroes in South Africa and the United States. Also, a marked difference is evident in persons in North and South India.

Ulcer is regarded as a possible stress disease and the increasing wear and tear of life in this age of rush, hurry, worry and frustrations may bear some relationship to ulcer formation and chronicity.

Our studies indicate that duodenal ulcer is more frequent in males and it may be interesting to note that the sex disparity may be hormonal in origin and that after the menopause, ulcers are about equal in both sexes. Ulcer may appear in several members of a family and the age of onset of ulcer in different sites shows a small but significant difference.

Ulcer in childhood has not been explained satisfactorily and there are many cases on record of ulcer in babies after normal unassisted delivery or delivery by Caesarian section. Ulcer, especially duodenal, occurs in older children and the physician must not minimize complaints referable to the stomach. Hemorrhage and perforation of an unrecognized ulcer in a child may be fatal.

In 1842, Curling called attention to the formation of ulcer in the duodenum after severe body burns. This complication is now rarely seen because of improved medical and antibiotic treatment given to patients following burns. Curling's ulcer occurs mainly in children; nearly half of them under the age of ten and most of the reported cases have been in girls, probably owing to the frequency with which girl's clothing catches fire. The ulcer is nearly always situated in the duodenum immediately beyond the pylorus and is generally of large size, rounded or irregular in shape and deeply excavated. It ends fatally, either by hemorrhage or perforation. Modern conditions of treatment may prevent and cause disappearance of the ulcer as a complication of burns.

Patients who complain of long standing dyspepsia, or heartburn should undergo thorough physical and gastrointestinal studies to rule out gastric or duodenal ulcer or gallbladder disease. Even if no direct evidence is found, they are to be cautioned not to over expose their bodies to the sun to a degree where a severe sunburn may result. Patients with known ulcers are also to avoid sunburn because activation of the ulcer may be the result, even if they follow the outlined treatment for ulcer.

Several patients, who during the summer months received more than superficial skin tanning, had recurring ulcer symptoms even though they were not in the "R" months. Others who went south during the winter and exposed themselves unduly to the solar rays, developed increased hyperacidity and aggravation of their ulcer.

SAMUEL WEISS, M.D.

BIBLIOGRAPHY

Cruveilhier, J., Anatomie Pathologique du Corps Human ,Bailliere, Paris, 1929, 1935.
Illingworth, C. F. W., Peptic Ulcer, E. S. Livington Ltd., Edinburgh & London, 1953.
Sandweiss, D. J., Peptic Ulcer, W. B. Saunders, Philadelphia, 1951.
Smith, Lucian, A., Peptic Ulcer, Appleton-Century-Crofts, Inc. ,New York, N. Y., 1953.

In Memoriam

We record with profound sorrow the passing of Dr. William E. Jones, Texarkana, Texas, Fellow and Dr. Martin Friedrich, Brooklyn, N. Y., Fellow, of the American College of Gastroenterology.

We extend our deepest sympathy to the bereaved families.

ABSTRACTS FOR GASTROENTEROLOGISTS

INTESTINES

INTESTINAL FUNCTION IN PATIENTS WITH ILEOSTOMY: Alvin J. Cummins. Am. J. Med. 16:237-245, (Feb.), 1954.

Observations are reported on the small bowel function of five patients with ileostomies studied by direct visualization and retrograde intubation. Emotional stress produced ileal hypermotility, hyperemia, and mucosal hyperfragility, demonstrating the participation of the small bowel in the response to stressful life situations previously documented for the stomach and colon. Eating, or in some cases, the anticipation of food, likewise resulted in ileal hyperfunction. The effect of anticholinergic drugs in modifying these as to the mechanism involved and then clinical and physiologic significance are discussed.

J. R. VAN DYNE

INDICATIONS FOR THE DOUBLE CONTRAST COLON EXAMINATION: C. A. Stevenson and M. Wilson. Am. J. Roentgenol, 71:398, (March), 1954.

In 1,000 patients the examination with the double contrast method revealed unsuspected polyps in only 0.4 per cent. Since the ascending colon was inadequately visualized in 20 per cent of these patients, it was apparent that the double contrast examination should not be used as a routine procedure because of the possibility of missing a lesion of the terminal ileum, cecum, or ascending colon. When the referring physician indicates the possibility of disease of the terminal ileum, right colon, or inflammatory disease, then complete roentgenoscopy, and roentgenograms before and after evacuation are indicated. All other patients may be examined by the double contrast method but it is necessary to adequately visualize the cecum and ascending colon. Re-examination will be necessary in about 20 per cent. As far as polyps are concerned, the double contrast examination will produce a diagnostic yield of about 5 per cent in all patients who have any type of rectal bleeding, polyps found at proctoscopic examination, or a history of polyps previously found or removed.

FRANZ J. LUST

ROENTGEN FINDINGS IN STRANGULATING OBSTRUCTIONS OF THE SMALL INTESTINE: Harry Z. Melline and Leo G. Rigler. Am. J. Roentgenol, 71:404, (March), 1954.

The earlier diagnosis of strangulating obstructions represents one of the most effective ways of lowering the current mortality rate of small intestinal obstruction. Roentgenologic evidence of strangulating obstruction can assist very appreciably in the early diagnosis. The roentgen signs are dependent upon the presence of a closed loop of intestine distended by either gas or fluid giving evidence of a fixed position. Twenty-six cases are reported, the following signs should lead to a diagnosis: 1) The coffee-bean shadow. 2) The "pseudo-tumor" shadow. 3) Fixation of the loop of intestine.

4) Loss of the normal mucous membrane pattern within the closed loop or above. Additional signs of collateral value are: 5) Absence of small intestinal gas in a case suspected clinically of small intestinal obstruction. 6) The presence of unusually large amounts of fluid in the lumen of the small intestine. 7) Long fluid levels far beyond the usual size. 8) Distention of a segment of the intestine far out of proportion to the remaining loops. 9) Absence of decompression of a localized loop following suction siphonage. 10) The presence of moderate amounts of gas in the colon despite the apparent evidences of small intestinal obstruction.

FRANZ J. LUST

ASSOCIATED JEJUNAL AND DUODENAL ULCERS: REPORT OF TWO CASES: K. J. Keeley. South African M. J., 28:212-213, (March 13th), 1954.

These two cases are unique, as each exhibited both jejunal and duodenal ulceration, in spite of there having been no previous surgery of the gastrointestinal tract. Both patients, who were respectively 42 and 50 years of age, complained of intractable epigastric pain of from 3 to 6 months' duration. Ascites was present in one and suspected in the second. One had severe bouts of diarrhea. Laparotomy in one patient uncovered a duodenal ulcer just distal to the pylorus which had involved the pancreas following perforation. Two inches beyond the beginning of the jejunum, a second ulcer was found which likewise had penetrated through the intestinal wall to become sealed off by the transverse mesocolon. Laparotomy was required for pyloric stenoses in the second patient and an ulcer was found in the first portion of the duodenum with a serious stricture of the lumen. Just distal to the duodenaljejunal junction another ulcer was present. This ulcer too seemed to have penetrated into the mesentery. It is felt that the jejunal ulcers are peptic in origin.

REGINALD B. WEILER

THE ACUTE STAPHYLOCOCCIC ENTEROCOLITIS OF ANTIBIOTIC THERAPY: G. Bickel and P. Rentchnick. Schweiz. med. Wchnschr., 84:311 (March 13), 1954.

This entity has been recently observed with increasing frequency after treatment with various antibiotics usually given for extraintestinal diseases. Symptoms appear on the third to sixth day after antibiotics have been started. Some cases show more a choleriform picture with intractable diarrhea, others exhibit a toxic reaction with sudden shock. Diagnosis is confirmed by the presence of a pure culture of staphylococci in the stool which are resistant to the previously administered antibiotics. Early diagnosis of this syndrome is mandatory. It may be suspected by the reappearance of fever, hypotension, and odorless watery stools. Treatment consists of maintenance of water and electrolyte balance with infusions, correction of shock with plasma, cortisone and ACTH and combating the superimposed staphylococcic infection with large doses of erythromycin, or other therapeutic agents toward which no resistance has been established.

H. B. EISENSTADT

POLYPS OF THE COLON AND RECTUM: J. Alfred Rider, Joseph B. Kirsner, Hugo C. Moeller and Walter L. Palmer. Am. J. Med., 16:555, (April), 1954.

The incidence of polyps in the rectum and the colon and their relationship to carcinoma was investigated in 400 patients with polyps. The cases were collected from a total number of 7,500 patients and represented an incidence of 5.4 per cent. In 93 per cent the diagnosis was made by rectoscopy, in 7 per cent by x-ray examination. The majority of polyps were found in the distal 25 centimeters of the large bowel. About half of the patients were asymptomatic, others complained of rectal bleeding, abdominal cramps and change of bowel habits. A patient who had one polyp was almost four times and a patient with several polyps eight times as prone to new polyp formation as the average patient attending a gastrointestinal clinic. There was no conclusive direct evidence of a transition of a benign polyp to a carcinoma with the passage of time. Newly discovered polypi of patients who were under observation for years were either benign or malignant from the beginning. Sometimes, however, one area of the polypus appeared benign while another was malignant under the microscopic examination. The evidence of carcinoma in the 400 polyp patients however was five

times greater than in the nonpolyp patients. The incidence of cancer in patients with several polyps was still greater. Local excision of benign polyps did not increase the likelihood of malignant transformation.

H. B. EISENSTADT

RECTAL AND COLONIC POLYPS: N. W. Swinton. Postgraduate Med. 15:451-458, (May), 1954.

The majority of benign rectal and colonic polyps are symptomless and do not hemorrhage. Routine sigmoidoscopy or x-ray is the usual mode of discovering these lesions. Such diagnostic procedures are mandatory in every complete examination. It is generally recognized that mucosal polyps are considered to be premalignant in nature. Surgical removal is the best therapy.

REGINALD B. WEILER

JEJUNAL ULCER: Samuel F. Marshall. Postgraduate Med. 15:527-531, (June), 1954.

Recurrent ulcers of the jejunum may follow surgery in which the small bowel is sutured to the stomach. Often hemorrhage is the first symptom of jejunal ulceration, and this is more common in this condition than ulceration elsewhere in the small bowel. Persistent high acid content and insufficient gastric resection, especially lack of removal of the antrum, are two etiological factors in recurrent jejunal lesions. Further, smoking, drinking, or dietetic indiscretions are provocative of jejunal ulcers following surgery higher in the tract. The author favors transabdominal vagotomy for jejunal ulcers where adequate previous resection has been performed.

REGINALD B. WEILER

DUODENAL DIVERTICULA—JEJUNOSTOMY: R. Robert De Nicola. Postgraduate Med. 15:489-492, (June), 1954.

Diverticula of the duodenum rarely becomes symptomatic unless they are large, fill easily and remain distended. Previously described surgical methods seem unnecessarily radical. Duodenal diverticulo-jejunostomy is offered as an alternate procedure in cases where excision seems to be unavailable.

The main practical consideration of a duodenal diverticulum is to prevent filling and retention in the pouch. Excision of the sac should be done in most cases but is not a "sine qua non" as the symptoms are not due to the sac "per se" but rather to its inability to empty itself. When the diverticulum is close to the ampulla of Vater excision is not always a safe procedure.

In as much as jejunal diverticula rarely cause trouble, the author's method, in cases where excision is inadvisable, is to anastomose it to the proximal jejunum. This relieves the symptoms without the risk involved of more extensive surgery. The duodenal coil and biliary tract are left in normal relationships and danger of compression in either system is avoided. The serosal covering of the diverticulum forms an excellent junction with the jejunum. However, perianastomotic sutures will further safeguard the anastomosis and serve to relieve the diverticulum of the drag from the jejunum.

REGINALD B. WEILER

EXPERIMENTAL STUDIES ON THE PATHOGENESIS OF NONSPECIFIC LOCALIZED ENTERITIS: Ryohei Okada. Nagoya Med. J. 1:193, (July), 1954.

The ileum of the rabbit, tested with coli filtrate and celomic fluid and extract of ascaris, showed positive Shwartzman phenomenon. Further the reaction resembled the acute forms of nonspecific localized enteritis.

B. Coli is found universally in the intestine and ascariasis is very common among oriental people. Hence it is natural that in the intestine there exist the active principles of Shwartzman phenomenon. When the active principles are absorbed through the intestinal wall, the intestine is in a preparatory state for Shwartzman phenomenon. In ascariasis, the mucosa is injured by the ascaris, and the absorption is made easier. When the active principles of the B. coli and the ascaris are absorbed into the blood stream, Shwartzman phenomenon, in other words nonspecific localized enteritis, will result. Further, in Shwartzman phenomenon, the allergic antigen antibody reaction acts as provocative injection, and hence, it is conceivable that the cause of this disease is rendered much easier.

FRANZ J. LUST

LIVER AND BILIARY TRACT

HEPATIC INSUFFICIENCY: Clinico-pathologic Conference (Washington Univ. School of Medicine). Am. J. Med. 16:272, (Feb.), 1954.

An unusual case of chronic hepatic failure due to the Chiari syndrome is discussed. The latter author described the syndrome bearing his name of chronic progressive hepatomegaly with ascites due to an obstructive disease of the hepatic veins. The large hepatic veins or the fine venous radicals might be involved in a process of thrombosis, thrombophlebitis or endophlebitis. The patient under discussion was a 17-year old female, suffering from fatigue, weakness, nausea and weight loss over a period of months and developing an unusual stubborn ascites with enlargement of the collateral veins of the abdominal as well as the thoracic wall. There were various abnormalities of the liver function tests, but a fairly normal serum protein and albumin/globulin ratio. The clinical picture was complicated by a progressing low salt syndrome terminating in a state of shock with death. Autopsy revealed an unusual form of hepatic cirrhosis with a combination of numerous thrombi in many small radicals of the hepatic vein with an extreme congestion and centrolobular atrophy of the liver cells and nodules of regeneration of the hepatic parenchyma. The picture appeared to be a combination of advanced cardiac and Laennec's cirrhosis. Small thrombi were present in the large hepatic veins and even in the inferior vena cava. The thrombi appeared to be at least as old or older than the parenchymal changes and were thought to be the cause of the liver cirrhosis. The origin of the thrombotic processes remained obscure but it might have been a primary inflammatory disease of the liver especially as the patient had infectious mononucleosis 4 years prior to the onset of the hepatic disease.

H. B. EISENSTADT

HEPATOLENTICULAR DEGENERATION. A NEW METABOLIC AFFECTION: G. Boudin and B. Pepin. Presse medicale, 62:243, (Feb. 17), 1954.

This disorder has three principal manifestations: hepatic cirrhosis, cerebral disturbances, which appear either in the form of extra-pyramidal hypertonia or cerebrellostriate hyperkinesia, and the Kayser-Fleischer ring of the cornea. In addition, psychotic manifestations, epilepsy, brownish-skin discoloration, diabetes mellitus, hypoglycemia, osteoporosis, spontaneous fractures, anemia, leukopenia, thrombocytopenia, albuminuria, cylindruria and azotemia have occasionally been present. In more than 50 per cent of the cases the disease is familial. The inheritance is recessive.

Two metabolic abnormalities are constantly present in this disorder. At first an excessive urinary excretion of all normal plasma amino acids; which is assumingly caused by a faulty renal reabsorption. Secondly, an abnormal retention of copper in various tissues, especially in those showing clinical abnormalities as the liver, the brain and the cornea. This increase of tissue copper is usually associated with an increase of plasma copper and a decrease of plasmatic caeruloplasmin, a normal serum alpha globulin. The logical treatment for the accumulation of copper in the body appears to be a prolonged administration of BAL. Such therapy has only a partial and temporary effect on the disease.

H. B. EISENSTADT

FRONT AND SIDE VIEW CHOLECYSTOGRAPHY WITH ACCELERATED AND TIMED EVACUATION. (F.P.A. CHOLECYSTOGRAPHY).-ALBOT-BUSSON-TOULET METHOD: G. Albot, A. Busson, J. Toulet and C. Cinqualbre. La Semaine des Hopitaux, 30: no. 16, (March), 1954.

F.P.A. cholecystography represents a very exact radiological method of exploring the functioning of the gallbladder while enabling its main features to be studied singly.

The examination is carried out in dorsal decubitus and front and side view exposures are made at each juncture by changing the position of the bulb but without moving the patient: after preparation, 5 minutes after Boyden's meal preceded by the ingestion of a 100 c.c. iced solution of sodium chloride at 7 per cent, 15 and 30 mins. after.

The semiological interpretation of the exposures enables the exact morphology of the gallbladder and the infundibulo-cystic

region to be appreciated at this point. The initial tonus of the gallbladder (in proportion to the vesicular-vertebral angle seen in side view at the start) can be measured; the effort of contraction can be calculated by measurement of the angle of vesicular erection in side view and a chronometrical graph can be established from it, exact calculation of volume (by J. Toulet's method) to be made and an exact chronovolumetric curve to be plotted. An appreciation of the variation in the impregnation of the choledoch which all depend on the functioning of the gallbladder is possible.

Comparison of these different elements facilitates very precise radiological diagnosis.

Contraction and evacuation graphs running parallel and of normal intensity for nonpathological gallbladders, may be in disagreement in cases of cystic obstruction (exaggerated contraction, insufficient evacuation).

There is the same appearance with hyposystolia, but the violent effort of contraction is generally temporary, only discernible on exposures made between the 5th and 15th minute and disappearing later. The two curves may remain parallel and in proportion to one another while both being insufficient (in hypotension) or on the contrary both exaggerated (in irritation of the gallbladder).

Finally, certain atonies of the sphincter are accompanied by feeble contractions, paradoxically associated with hyperevacuation.

By this method the main biliary duct is normally impregnated between 5 and 15 minutes after Boyden's meal. Shown up very clearly at the 5th minute in cases of irritation of the gallbladder, the choledoch is on the contrary invisible during the whole examination in cases of cystic obstruction. Its impregnation may be slight, retarded (from the 15th minute only) and prolonged (still clear at the 30th minute) in vesicular hypotension and slight cystic obstruction.

F.P.A. cholecystography at present appears to be the most exact method for preoperative diagnosis in most patients and should limit still further the use of biliary radiomanometry for diagnosis leaving it however its indispensable role in pre- and postoperative control.

THE RADIOLOGICAL SYNDROME OF HYPEREVACUATION OF THE GALLBLADDER: Guy Albot, Jacques Toulet and Madame H. Treheux. La Semaine des Hopitaux, 30: no. 16, (March), 1954.

Front and side-view cholecystographies and the exact calculation of the coefficient of the volume of vesicular evacuation have enabled the writers to observe in numerous cases a radiological syndrome of vesicular hyperevacuation. Contrary to the opinion universally held until now, it is a question not of normal gallbladders "very well emptied", but of pathological gallbladders "emptied too much".

The most typical syndrome consists of an initial normal tension or hypertension, marked hyperkinesia, hyperevacuation and the early appearance of the biliary ducts. It is therefore contrary to the present well-known syndromes of atony, vesicular hypotension, obstruction of the gallbladder with hypertonic vesicular stasis, mechanical asystolia by distention of the gallbladder behind a cystic obstruction.

These pathological disturbances in the functioning of the gallbladder, of the hyperevacuation type, may be purely functional and of extremely common occurrence during affections far removed from the digestive tract (duodenal ulcer, duodenitis, spasmodic colitis) and their interest is then mainly documentary.

This functional cholecystographic syndrome occurs in certain conditions of true migraine where it is of indisputable therapeutic interest.

Finally, during certain diffuse organic disorders of the gallbladder, which are dysplasic or inflammatory in nature, the same syndrome may be observed, this time organic or organo-functional, revealing diffuse irritation of the gallbladder; it may later turn out to be just the opposite syndrome already described by the writers under the name of early inflammatory asystolia of the gallbladder.

The study of irritable gallbladders, the comparison of the results obtained by accelerated front and side view cholecystography and by timed duodenal tubing pose physiopathological problems which are sometimes difficult to solve but, for which complementary explorations, in particular cholecystographic tubing, enable satisfactory explanations to be given although these are very different from formerly accepted notions. The writers believe the duodenal spasms sometimes play a part in anomalies

of bile flow during timed tubing, they indicate that hyperconcentration of the bile B may result from hyperactivity of the mucous membrane of the gallbladder parallel to a motor hyperactivity of its wall in the case of a hyperevacuated gallbladder.

THE MECHANISM OF ASCITES: Robert E. Hyatt and John R. Smith. Am. J. Med., 16: No. 3, (March), 1954.

Liver disease and congestion of hepatic and portal venous systems are the disorders most frequently associated with ascites. A correlation of the various factors contributing to this abnormal fluid accumulation is attempted in the light of modern laboratory experiments. Ascites is produced by a constriction of the inferior vena cava above the hepatic veins. This causes the accumulation of the large amount of fluid high in protein in the peritoneal cavity. The same change occurs after embolic or toxic injury of the liver. Constriction of the portal vein alone does not produce ascites in animals. The influence of portal obstruction in humans is difficult to assess as there is little correlation between the elevation of portal pressure and the amount of fluid accumulation. Similarly, no constant relationship exists between hypoproteinemia and ascites. As the peritoneal fluid is rich in protein one might expect that the oncotic pressure of this substance contributes to the perpetuation of the ascites. Actually, the fluid volume of the abdomen remains constant or even decreases after the intraperitoneal injection of salt-poor albumin. Much more important is the sodium retention accompanying ascitic states caused by increased tubular reabsorption. Salt retention is influenced by volume and osmotic receptors and adrenocortical and antidiuretic hormones. None of the above mentioned changes explains the origin of ascites. Therefore, the author assumes, on the basis of animal experiments, that this fluid is derived directly from an out-pouring of the hepatic lymph through the liver capsule. This would explain best the high protein contents and the observation that ascites formation precedes sodium retention. Once the fluid is accumulated in the abdomen the homeostatic mechanism of the body is activated setting up an equilibrium between the ascites and the normal fluid compartments. This leads to the hormonal and renal functional changes with sodium retention that perpetuate the ascites. The new fluid space is by no means static. Its protein content is completely turned over every few days, its water is totally exchanged every few hours. Therapeutic considerations derived from these investigations are that the primary objective of treatment must be the restoration of the liver fuction and the removal of the hepatic congestion. Less important, but also efective, is sodium restriction. All other therapeutic efforts have failed.

H. B. Eisenstadt

PATHOLOGY AND LABORATORY RESEARCH

AN ASSAY OF THE PHYSIOPATHOLOGY OF TIMED INTUBATION OF THE DUODENUM: G. Albot, M. Kapandji and M. Dressler. Arch. Mal. app. digestif. 43:5, 1954.

The authors bring evidence showing the role played by the duodenum in the prolongation of the closure period of the sphincter of Oddi and in anomalies of the flow of the bile B and their evidence is likely to cause a revision of the physiopathology of duodenal intubation.

The case in question was that of a woman suffering from painful attacks in the right hypochondrium and to whom were administered one after the other 2 cholecystographies, 2 timed duodenal intubations, 1 timed cholecystographic intubation and a pre- and postoperative biliary radiomanometry. The cholecystography showed a hyperkinetic gallbladder emptying to 75 per cent and the biliary radiomanometry enabled the existence of irritation of the gallbladder with feeble spasms of the cystic duct and of the sphincter of Oddi to be diagnosed.

Timed intubation of the duodenum on the other hand, interpreted by the usual standards, would have suggested dyskinesia of the cystic duct with hypertension of the sphincter of Oddi: during the intubation there was observed a 10 minute period of closure of the sphincter of Oddi giving way after ingestion of novocaine which brings on an immediate flow of hyperconcentrated bile B; a second intubation was marked by a 10 minute phase during which the sphincter

of Oddi was closed, a prolonged vesicular phase of 45 minutes with hyperconcentrated bile B.

Now cholecystographic intubation has enabled the apparent contradiction existing between cholecystography and radiomanometry on the one hand and timed intubation on the other to be resolved.

Considering cholecystographic intubation without the radiographic exposures made during its course, results were noted comparable to the previous intubations, the sphincter of Oddi closed for 20 minutes followed by a very long vesicular phase of 65 minutes with interrupted flow, a period during which there was pain in the right side of the hypochondrium.

If this same intubation were interpreted in the light of exposures made during its administration it would be noted:

1) That during the period that the sphincter of Oddi is closed for an abnormal length of time (20 minutes) the volume of the gallbladder diminished from 18 to 15 cm, that the cystic and the choledoch became suffused and bile from the gallbladder colored with Telepaque passed into the duodenum 10 minutes after the beginning of the closed sphincter phase but that this bile was only visible in the upper portion of the duodenum and was not in contact with the bulge in the probe.

2) That during this time there existed a medioduodenal spasm, as could be deduced from the fact that the part of the probe in the second portion of the duodenum came near the vertebral column becoming rectilinear instead of concave, and that the bile accumulated in the proximal superior pouch of the second part of the duodenum without coming into contact with the bulge of the probe in the distal pouch at the *genu inferius*.

3) That the gallbladder became invisible and consequently almost empty 15 minutes after the beginning of the vesicular phase whilst the flow of bile B through the probe was prolonged for 65 minutes. It may thereby be concluded that in reality the emptying of the gallbladder was very much more rapid than the intubation led to suppose; the flow which followed the radiological emptying of the gallbladder was that of the bile B stored in the proximal pouch of the duodenum and mixed with the hepatic bile which arrived in the distal pouch after reduction of the medioduodenal spasm.

4) That furthermore, the interruption of the flow of bile was not necessarily the result of disturbances of the contraction and emptying of the gallbladder since this irregularity in the flow was produced during the evacuation of the bile stored in the duodenum.

The authors conclude that in timed duodenal intubation the role of the duodenum is likely to modify completely the significance of the results of the intubation. They believe it necessary to carry out timed intubation of the duodenum with the help of a double coupled probe whose end openings correspond respectively with each of the two duodenal pouches in case of medioduodenal spasm.

EFFICACY AND TOXICITY OF OXYTETRACYCLINE (TERRAMYCIN) AND CHLORTETRACYCLINE (AUREOMYCIN). Maxwell Finland, Margaret E. Grigsby and Thomas H. Haight. A.M.A. Arch. Int. Med., 93:23-43, (Jan.), 1954.

Five hundred twenty patients with various infectious diseases, mostly of the respiratory or urinary tract were treated with oxytetracycline (Terramycin) or chlortetracycline (Aureomycin). The frequency of gastrointestinal complications, particularly of watery diarrhea, was significantly greater in persons treated with Terramycin than with Aureomycin. The toxic side-effects could be considerably reduced by restricting the oral dosage of these substances to 1 gram in 24 hours. This amount was sufficient for the majority of cases but had to be supplemented in severe infections by intravenous dosage. Coagulase positive staphylococci were frequently found as the only or the predominant intestinal organism after oral usage of both antibiotics. Their importance, however, as causative organisms of the diarrheal disturbances remains to be established. The tendency to serious diarrheas following oral antibiotics is increased by the intake of any laxative which, therefore, should be completely avoided. The best treatment for such diarrheas is immediate cessation of the administration of the offending substances together with supportive measurements as proper hydration and electrolyte replacement. This is more important than the administration of other antibiotics as erythromycin, neomycin or bacitracin. The effectiveness of these substances is unproven. The rapid return of the normal flora after simple supportive treatment is noteworthy.

H. B. EISENSTADT

POTENTIALLY REVERSIBLE RENAL FAILURE FOLLOWING EXCESSIVE CALCIUM AND ALKALI INTAKE IN PEPTIC ULCER THERAPY: Francis X. Dufault, Jr. and G. James Tobias. Am. J. Med., 16:231, (Feb.), 1954.

The danger of producing systemic alkalosis and renal dysfunction by prolonged intake of absorbable alkali was first described by Hard and Rivers in 1923. Such abnormalities, however, have been recently called Burnett syndrome because Burnett, et al, directed the attention to the associated nephrocalcinosis and metastatic calcification. Four cases with this syndrome are described who developed muscular weakness, fatigue, anorexia, nausea, vomiting, weight loss, constipation, polydipsia, polyuria and pruritus due to hypercalcemia. The existing nephropathy was characterized by azotemia, isosthenuria, delayed P.S.P. elimination, albuminuria and presence of casts and red and white cells in the sediment. Apart from the history of a chronic peptic ulcer and the excessive intake of absorbable alkali for years, the entity was suspected by the presence of calcifications in the cornea and conjunctiva. The excessive intake of calcium as well as of sodium bicarbonate or other absorbable alkali can produce nephrotubular damage with the same syndrome. Treatment consists of low calcium diet and avoidance of absorabable alkali. Whenever a chronic pyelonephritis is superimposed antimycotic therapy has to be added.

H. B. Eisenstadt

PREFRONTAL INFILTRATION OF NOVOCAINE IN THE PATHOLOGY OF THE DIGESTIVE SYSTEM: R. Cattan, P. Frumusan, M. Bucaille, A. Abaza and C. Zaidmann. Arch. Mal. app. Dig. 43: No. 3, (Feb.), 1954.

In the hope of bringing relief to sufferers from incurable cancers, M. Bucaille perfected two years ago a method which, by using special apparatus and exact radiological location, enables a very tiny portion of the prefrontal lobe to be infiltrated. The substance used is a 1 per cent solution of novocaine.

The authors have used this method in three cases of very serious digestive hemorrhage and in one of gastric ulcer. Hemorrhage was due in the first case to a diverticulosis of the sigmoid flexure in a patient suffering from progressive rheumatoid arthritis, and in the second to an ulcerative rectocolitis, in the third to a latent duodenal ulcer. In the three cases, infiltration caused an immediate cessation of the hemorrhage. Furthermore, the progressive rheumatoid arthritis of the first patient was considerably improved. The very serious attack of ulcerative colitis was mitigated but there was a relapse, benign in nature, a few months later.

The gastric ulcer was an outsize ulcer of the lesser curvature in an old man. Very varied medical treatments which had been tried for three months had failed to relieve the intolerable pains. Bilateral infiltration of the prefrontal lobe caused the immediate disappearance of pain and a rapid cure of the niche.

The authors discuss the working of the method which cuts, by dilaceration, the fibers leading from the frontal cortex to the thalamus. The grey matter being hardly touched, no personality change nor any of the disadvantages of lobotomy are observed. These cases throw new light on the well-known action of psychism in some affections of the digestive tract. These should be studied together with the psychosomatic and corticovisceral theories at present held in various countries.

PSYCHOSOMATIC MEDICINE

AN EVALUATION OF HYPNOTICALLY INDUCED RELAXATION FOR THE REDUCTION OF PEPTIC ULCER SYMPTOMS: Hamilton Moady. Brit. J. Med. Hypnotism. 5:23-33, 1953.

An attempt was made to prove that hypnotic relaxation would reduce the symptoms of duodenal ulcer. This "appears to have been proved" by x-ray. Secondary criteria "were the self-evaluation of the patient" and the "physician's medical progress notes". The patient's evaluations "led to the impression that clinical improvement was greater than had been indicated by the radiographic ratings". The improvement is "due to experimental hypnotherapy".

Reginald B. Weiler

FOOD ALLERGIES AND CONVERSION HYSTERIA: Melitta Sperling. Psychoanalyt. Quart. 22:525-538, 1953.

Although symbolism of food is well recognized, such symbolism in relationship to *specific* allergies is less well investigated. It is important to recognize the place of dynamic potential energy, possessed by unconscious fantasies, as being a factor in the genesis of somatic symptoms. When repressed material threatens to become conscious, anxiety was experienced but no physical symptom became manifest. Previous to analysis, frustration or temptations produced somatic symptoms but no anxiety. Later, when the unconscious meanings were recognized, neither symptoms nor anxiety were felt. Therefore, it seems that when a threatened breakdown of defenses is imminent, with conscious control lacking, conversion of affects and fantasies into specific somatic expressions takes place.

Anal eroticism is significant, as a specific genetic and dynamic factor in allergic conditions. In the psychoanalysis of allergies of the skin, respiratory, or gastrointestinal systems there is a close connection between the reactions and pregenital, unconscious impulses and fantasies; so that allergic symptoms may be somatic equivalents of latent perversions.

REGINALD B. WEILER

SHORT-TERM PSYCHOTHERAPY OF A CASE OF CONVERSION HYSTERIA: H. Stekel. Am. J. Psychotherapy. 7:302-309, 1953.

A 34-year-old homosexual man suffered from attacks of dyspnea and painful dysphagia. After 25 weekly sessions the symptoms were removed. It was determined that the attacks were precipitated by identification with his dying father whose dyspnea was reflected in the patient's attacks. The dyspnea and dysphagia also represented defenses against his sadomasochistic and homosexual drives.

REGINALD B. WEILER

Easy as a.b.c...

the FLEET ENEMA

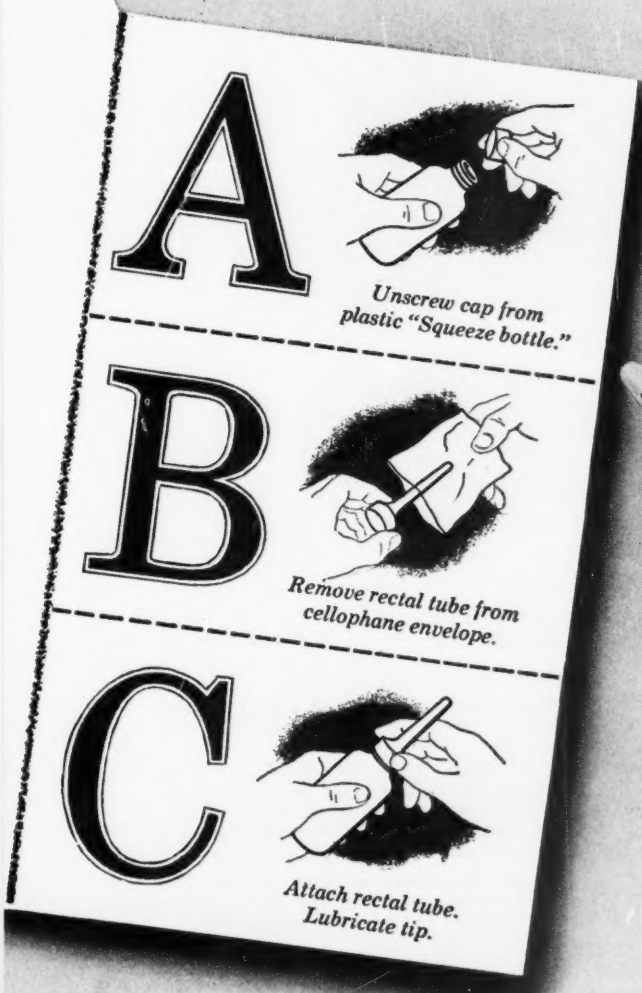
Disposable Unit

Polyethylene "squeeze bottle" permits easy one-hand administration... rectal tube kept sanitary by sealed cellophane envelope... distinctive rubber diaphragm prevents leakage while controlling flow. Because of these unique features, FLEET ENEMA Disposable Unit is preferred for hospital, clinic and office use.

Each single use unit of 4½ fl. ozs., contains in each 100 cc., 16 Gm. sodium biphosphate and 6 Gm. sodium phosphate,.. an enema solution of Phospho-Soda (Fleet), gentle, prompt, thorough—and as effective as the average enema of one or two pints.

C. B. FLEET CO., INC.
Lynchburg • Virginia

'Phospho-Soda' and 'Fleet' are registered trademarks of C. B. Fleet Co., Inc.



in this moment

The prayer lingers still . . . across the table as Dad begins to serve . . . it brushes Mother's still-bowed head . . . it caresses Sally's fist as she reaches for the promised drumstick. The words of thanksgiving are being made real in this moment—gratitude from a good provider to the Great Provider . . . in this time of security together.

The most precious gift we give or receive is the gift of security. Only in a land like ours are we free to choose security as a goal of living.

And through this choice we achieve another great gift. For, secure homes, one joining another, make up the security of America.

Saving for security is easy—on the Payroll Savings Plan for investing in United States Savings Bonds.

This is all you do. Go to your company's pay office, choose the amount you want to save—a few dollars a payday, or as much as you wish. That money will be set aside for you before you even draw your pay. And automatically invested in United States Series "E" Savings Bonds which are turned over to you.

If you can save only $3.75 a week on the Plan, in 9 years and 8 months you will have $2,137.30.

U.S. Series "E" Savings Bonds earn interest at an average of 3% per year, compounded semiannually, when held to maturity! They can go on earning interest for as long as 19 years and 8 months if you wish.

If you want interest as current income **ask your bank about 3% Series "H" Bonds which pay interest semiannually by Treasury check.**

The U. S. Government does not pay for this advertisement. It is donated by this publication in cooperation with the Advertising Council and the Magazine Publishers of America.

newest

broad-spectrum antibiotic

Tetracyn*

Brand of **tetracycline**

is now available on prescription from *Pfizer Laboratories,* Division, Chas. Pfizer & Co., Inc., world's largest producer of antibiotics, discoverers of oxytetracycline and the first to describe the structure of tetracycline, nucleus of modern broad-spectrum antibiotic therapy.

Tetracyn is supplied as Capsules, Tablets, Oral Suspension (chocolate flavored), Pediatric Drops (banana flavored), Intravenous, Intramuscular, Ophthalmic Ointment, and Ointment (topical).

*TRADEMARK

For well-tolerated therapy of such common infections as:

Pneumococcal infections, including pneumonia, with or without bacteremia; streptococcal infections, with or without bacteremia, including follicular tonsillitis, septic sore throat, scarlet fever, pharyngitis, cellulitis, urinary tract infections due to susceptible organisms, and meningitis; many staphylococcal infections, with or without bacteremia, including furunculosis, septicemia, abscesses, impetigo, acute otitis media, ophthalmic infections, susceptible urinary tract infections, bronchopulmonary infections, acute bronchitis, pharyngitis, laryngotracheitis, tracheobronchitis, sinusitis, tonsillitis, otitis media, and osteomyelitis; certain mixed bacterial infections; soft tissue infections due to susceptible organisms.

Pfizer PFIZER LABORATORIES, Brooklyn 6, N. Y.
Division, Chas. Pfizer & Co., Inc.

and the older antacid tablets just won't disintegrate!

now TREVIDAL® tablets

break up in less than a minute, assuring rapid yet prolonged relief of hyperacidity.

EACH TREVIDAL TABLET CONTAINS:

Balance of ingredients avoids constipation, diarrhea, or alkalosis →	Aluminum hydroxide gel, dried . . . 90 mg. Magnesium trisilicate . . . . . 150 mg. Magnesium carbonate . . . . . 60 mg. Calcium carbonate . . . . . 105 mg.
Unique vegetable mucin supplies protective coat to irritated stomach lining →	Regonol*† . . . . . 100 mg.
Binder controls and extends antacid activity →	Egraine* . . . . . 45 mg.

AVAILABLE IN BOXES OF 100 TABLETS, SPECIALLY STRIPPED FOR EASY CARRYING.

†Cyamopsis tetragonoloba gum
*Trade Marks

Organon INC. • ORANGE, N. J.

"complete symptomatic relief"[1] *in peptic ulcer patients...*

Antrenyl®

In a recent study, patients with acute symptoms of peptic ulcer obtained relief 24 to 36 hours after taking Antrenyl, a potent anti-ulcer agent.

ANTRENYL—prescribed as an adjunct to rest, sedation, antacids and diet—offers the peptic ulcer patient optimal benefits. It is also of value in other conditions marked by gastrointestinal spasm.

ANTRENYL inhibits gastrointestinal motility and gastric secretion. Side effects are either mild or absent, and there is no bitter aftertaste.

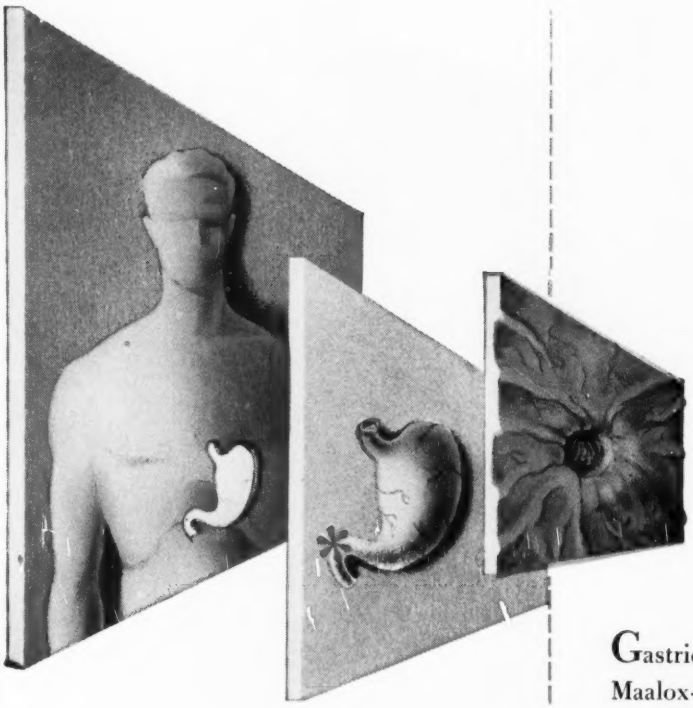
ANTRENYL is available as tablets (white, scored), 5 mg.; syrup, 5 mg. per 4-ml. teaspoonful; tablets (peach-colored, scored), 5 mg. with phenobarbital, 15 mg.; Pediatric Drops (with dropper), each drop containing 1 mg. of Antrenyl bromide.

1. ROGERS, M.P., AND GRAY, G.L.: AM. J. DIGEST. DIS. 19:180 (JUNE) 1952.

Antrenyl® bromide (oxyphenonium bromide CIBA)

CIBA
SUMMIT, N. J.

R/20321M



maalox®

gives ulcer relief

without side effects

Gastric hyperacidity is controlled by Maalox-Rorer without constipation or other side effects commonly encountered with antacids. Relief of pain and epigastric distress is prompt and long-lasting. Available in tablets and liquid form.

Suspension Maalox-Rorer contains th-hydroxides of Magnesium and Aluminum in colloidal form. The smooth texture and pleasant flavor make it highly acceptable, even with prolonged use.

Supplied: in 355 cc. (12 fluid ounce) bottles. Also in bottles of 100 tablets. (Each Maalox tablet is equivalent to 1 fluidram of Suspension Maalox.)

Samples will be sent promptly on request.

WILLIAM H. RORER, INC.

Drexel Bldg., Independence Square

Philadelphia 6, Pa.

for the patient
who balks
at
taking
hydrophilic
colloids

. . . prescribe

L.A. FORMULA

in milk or
orange juice

L.A. FORMULA

a bulk producer
unsurpassed
for
patient acceptance

L. A. FORMULA — *50% Plantago ovata concentrate dispersed in lactose and dextrose and refined to a unique particle size.*

Available—7 and 14 oz. containers. Samples on request.

BURTON, PARSONS & COMPANY • Washington 9, D.C.

". . . the gastric secretion is the immediate agent of mucosal tissue digestion. . . . Opposed to this stands the defensive factor . . . the two-component mucous barrier"[1] [*the protecting layer of mucus and the mucosal epithelium*].

Rotational gastroscopic views showing coating effect 1½ hours after administration of Amphojel.[2]

Causation — key to treatment in peptic ulcer

Through *topical* action alone, AMPHOJEL contends with the local causes of ulcer—aggressive acidity coupled with impairment of the wall defenses. Providing a dual approach, AMPHOJEL combines two aluminum hydroxide gels, one reactive, one demulcent. The reactive gel combats the attacking factor in ulcer by promptly buffering gastric acid. The demulcent gel promotes healing of the denuded mucosa by forming a viscous, protective coagulum.

AMPHOJEL—nonsystemic, nontoxic—provides time-proved *fundamental* therapy.

AMPHOJEL®

ALUMINUM HYDROXIDE GEL

Supplied: Liquid, bottles of 12 fluidounces
Tablets, 5 grain, boxes of 30, bottles of 100;
and 10 grain, boxes of 60 and 1000

References: 1. Hollander, F.: Arch. Int. Med. *93*:107 (Jan.) 1954
2. Deutsch, E.: Scientific Exhibit, Gastroscopy, Clinical Meeting A.M.A., St. Louis, December, 1953

Wyeth®
Philadelphia 2, Pa.